criteria for a recommended standard . . . .

OCCUPATIONAL EXPOSURE TO PHOSGENE

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
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PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace. The National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices, to provide relevant data from which valid criteria for effective standards can be derived. Recommended standards for occupational exposure, which are the result of this work, are based on the health effects of exposure. The Secretary of Labor will weigh these recommendations along with other considerations such as feasibility and means of implementation in developing regulatory standards.

It is intended to present successive reports as research and epidemiologic studies are completed and as sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on phosgene by members of my staff and the valuable constructive comments by the Review Consultants on Phosgene, by the ad hoc committees of the American Academy of Occupational Medicine and the American Academy of Industrial Hygiene, and by Robert B. O'Connor, M.D., NIOSH consultant in occupational medicine. The Department of the Army, Edgewood Arsenal, Aberdeen Proving Ground, Maryland, and the Ministry of Defence, Chemical Defence Establishment, Porton, England, have been very helpful in

declassifying documents so they could be reviewed for use in this report. The NIOSH recommendations for standards are not necessarily a consensus of all the consultants and professional societies that reviewed this criteria document on phosgene. Lists of the NIOSH Review Committee members and of the Review Consultants appear on the following pages.

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The Division of Criteria Documentation and Standards Development, National Institute for Occupational Safety and Health, had primary responsibility for development of the criteria and recommended standard for phosgene. The University of Washington, School of Public Health and Community Medicine, developed the basic information for consideration by NIOSH staff and consultants under contract No. HSM-99-73-36. Jon R. May, Ph.D., had NIOSH program responsibility, and Sonia Berg served as criteria manager.

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CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN OCCUPATIONAL EXPOSURE STANDARD FOR PHOSGENE

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I. RECOMMENDATIONS FOR A PHOSGENE STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that worker exposure to phosgene in the workplace be controlled by requiring compliance with the following sections. The standard is designed to protect the health and safety of workers for up to a 10-hour workday, 40-hour workweek over a working lifetime. Compliance with the standard should therefore prevent adverse effects of phosgene on the health and safety of workers. The standard is measurable by techniques that are valid, reproducible, and available to industry and government agencies. Sufficient technology exists to permit compliance with the recommended standard. The standard will be subject to review and revision as necessary.

"Phosgene" is defined as gaseous or liquified phosgene. Synonyms for phosgene include carbonyl chloride, carbon oxychloride, chloroformyl chloride, and CG (designation used by military agencies). "Occupational exposure to phosgene" is defined as exposure above half the recommended time-weighted average (TWA) environmental limit. Exposure at lower concentrations will not require adherence to the following Sections except for Sections 3, 4(a), 4(b), 4(c)(3), 4(c)(5), 4(c)(6), 5, 6, 7, and 8(a). "Overexposure" is defined as known or suspected exposure above either the TWA or ceiling concentrations, or any exposure which leads to development of pulmonary symptoms.

Section 1 - Environmental (Workplace Air)

(a) Concentration

Occupational exposure to phosgene shall be controlled so that no worker is exposed to phosgene at a concentration greater than one-tenth part phosgene per million parts of air (0.1 ppm) determined as a TWA concentration for up to a 10-hour workday, 40-hour workweek, or to more than two-tenths part phosgene per million parts of air (0.2 ppm) as a ceiling concentration for any 15-minute period.

(b) Sampling and Analysis

Procedures for sampling, calibration of equipment, and analysis of environmental samples shall be as provided in Appendices I and II, or by any method shown to be equivalent in precision, accuracy, and sensitivity to the methods specified.

Section 2 - Medical

(a) Comprehensive preplacement and annual medical examinations shall be made available to all workers to be occupationally exposed to phosgene unless a different frequency is indicated by professional medical judgment based on such factors as emergencies, variations in work periods, and preexisting health status of individual workers.

(b) These examinations shall include, but shall not be limited to:

(1) Comprehensive or interim medical and work histories.

(2) A comprehensive medical examination giving particular attention to pulmonary function. Preplacement and follow-up pulmonary function tests shall be performed and shall include the forced vital

capacity (FVC), the one-second forced expiratory volume (FEV 1), and the forced midexpiratory flow (FEF 25-75); a preplacement chest X-ray shall be obtained. The possibility of increased risk for workers with preexisting cardiovascular or pulmonary diseases should be considered and, when appropriate, the workers should be given counseling on the possibility of increased risk. Return to work after an absence for sickness due to phosgene overexposure shall require medical approval.

(3) A judgment of the worker's ability to use a negative or positive pressure respirator.

(c) Proper medical management shall be provided for workers overexposed to phosgene.

In case of known or suspected overexposure to phosgene, first aid measures shall be taken immediately, followed by prompt medical evaluation and care. Overexposed persons should not be permitted any unnecessary physical exertion. They should be carried to a vehicle for subsequent transportation to receive medical assistance. Pressurized oxygen and attendants trained in its use shall be available in the event they are needed for persons in respiratory distress. In case of skin or eye contact with liquid phosgene, contaminated clothing shall be removed immediately and the exposed body areas flushed with copious amounts of water. The plant physician or medical consultant shall be informed of any suspected overexposure to phosgene and shall determine the need for X-ray or pulmonary function studies or hospitalization. Because of the often-delayed onset of symptoms following overexposure to phosgene, surveillance or monitoring of the patient by a physician or by trained paramedical personnel is required for the 24-hour period following overexposure. A

posterior-anterior chest film should be taken in each instance of known or suspected overexposure to phosgene for comparison with preplacement chest films. Pulmonary function tests may be useful during convalescence.

(d) Medical records shall be maintained for all workers occupationally exposed to phosgene. All pertinent medical records with supporting documents, including chest films for at least the 5 years preceding termination of employment and the original preplacement chest films, shall be maintained for at least 5 years after the termination of the individual's employment. The designated medical representatives of the Secretary of Health, Education, and Welfare, of the Secretary of Labor, of the employer, and of the employee or former employee shall have access to these medical records.

Section 3 - Labeling (Posting)

All containers of phosgene and all areas where phosgene is stored, handled, used, or formed shall be labeled and placarded in accordance with An Identification System for Occupationally Hazardous Materials, a recommended standard published by NIOSH, and in accordance with the following subsections.

(a) Containers of phosgene shall bear the following label in addition to, or in combination with, labels required by other statutes, regulations, or ordinances:

CONTAINS PHOSGENE

EXTREME HEALTH HAZARD

Harmful or fatal if inhaled, may cause delayed lung injury.
Do not breathe gas.
Do not get liquid in eyes, on skin, or on clothing.
Use only with adequate ventilation and/or in closed systems.
Open containers with care.
Have respiratory protection available for emergency.

FIRST AID CALL A PHYSICIAN IMMEDIATELY

In case of inhalation, remove victim to uncontaminated atmosphere.
If breathing stops, administer artificial respiration.
Do not allow victim to walk or exercise.
In case of liquid contact, immediately flush skin or eyes with water.
Remove contaminated clothing without delay and dispose of liquid properly.

(b) The following warning sign shall be affixed in a readily visible location at or near entrances to areas in which phosgene is stored, handled, used, or formed:

CONTAINS PHOSGENE

EXTREME HEALTH HAZARD

Harmful or fatal if inhaled, may cause delayed lung injury.
In emergency, enter only if wearing respiratory, eye, and skin protection.
Phosgene respiratory protection located at (specific locations to be supplied by employer).
Unauthorized persons keep out.

This sign shall be printed both in English and in the predominant language of non-English-speaking workers, if any. All employees shall be trained and informed of the hazardous area with special instruction given to illiterate workers.

(c) All systems, piping, and associated equipment containing

phosgene shall be plainly marked for positive identification in accordance with American National Standard A13.1-1956. Shut-off valves shall be conspicuously labeled. Phosgene containers in use shall be plainly marked "In Use" to distinguish them from those not in use.

Section 4 - Personal Protective Equipment and Clothing

Engineering controls shall be used to maintain phosgene concentrations below the prescribed limits. When necessary, this shall be supplemented by the use of personal protective equipment. Requirements for personal protective equipment shall be in accordance with provisions of 29 CFR 1910 (Federal Register 39:23670, June 27, 1974).

(a) Skin Protection

(1) In addition to the respiratory protection specified in Table I-1, personnel performing emergency operations involving exposure to liquid phosgene shall wear one-piece suits, impervious to phosgene and tight at the ankles, wrists, and around the neck and face. The suits shall be ventilated with supplied air, preferably cooled, or time in the work area shall be limited with due consideration to the heat stress factors involved. Impervious gloves and boots shall also be worn. Such protective clothing shall be available at a convenient location outside the contaminated area.

(2) The employer shall insure a sufficient supply and adequate maintenance of protective clothing.

(b) Eye Protection

Personnel handling liquid phosgene in situations where eye contact can occur shall have eye protection afforded by full-face respiratory

protection as specified in Table I-1, since concentrations of phosgene sufficient to cause eye damage are also likely to cause respiratory tract damage.

(c) Respiratory Protection

(1) Compliance with the exposure limits may be achieved by the use of respirators only:

(A) during the time period necessary to install and test the controls required by Section 6(b) of this chapter;

(B) for nonroutine operations such as a brief exposure in excess of the TWA or ceiling concentration exposure limit as a result of maintenance or repair activities; or

(C) in emergencies when air concentrations of phosgene may exceed the TWA exposure limit.

(2) When a respirator is permitted by paragraph (1) of this subsection, it shall be selected from among those jointly approved by the Bureau of Mines, US Department of the Interior, and by the National Institute for Occupational Safety and Health, US Department of Health, Education, and Welfare, under the provisions of 30 CFR 11. The employer shall provide the respirator required and shall ensure its use. A respiratory protection program meeting the requirements of 29 CFR 1910.134, as amended, shall be established and enforced by the employer. Only appropriate respirators as described in Table I-1 shall be used.

TABLE I-1

RESPIRATOR SELECTION GUIDE

Air Concentrations	Respirator Type*
Less than or equal to 1 ppm	(1) Any supplied-air respirator; or (2) Any self-contained breathing apparatus.
Less than or equal to 2ppm	(1) Any supplied-air respirator with a full facepiece, helmet, or hood; or (2) Any self-contained breathing apparatus with a full facepiece.
Greater than 2 ppm or emergency situations	(1) Self-contained breathing apparatus with a full facepiece operated in pressure-demand or other positive pressure mode; or (2) A combination respirator which includes a Type C supplied-air respirator with a full facepiece operated in pressure-demand or other positive pressure or continuous flow mode and an auxiliary self-contained breathing apparatus operated in pressure-demand or other positive pressure mode.

TABLE I-1 (CONTINUED)

RESPIRATOR SELECTION GUIDE

Air Concentrations	Respirator Type*
Firefighting	Self-contained breathing apparatus with a full facepiece operated in pressure-demand or other positive pressure mode.
Evacuation or escape	(1) Any gas mask providing protection against phosgene; or (2) Any escape self-contained breathing apparatus with full facepiece.

* Approved by the Bureau of Mines and the National Institute for Occupational Safety and Health

(3) Each work area where there is potential for occupational exposure to phosgene shall have at least 2 sets of self-contained breathing apparatus readily available in nearby locations which do not require entry into, or passage through, a contaminated area for access.

(4) Respirators specified for use in higher concentrations of phosgene may be used in atmospheres of lower concentrations.

(5) Employees shall be trained and drilled in the use of

respirators assigned to them and in testing for leakage.

(6) Canisters shall be discarded and replaced with fresh canisters after use. Unused canisters shall be discarded and replaced when the seal is broken or when the shelf life, as recommended by the manufacturer, is exceeded.

Section 5 - Informing Employees of Hazards from Phosgene

At the beginning of employment, workers who will work in areas required to be posted in accordance with Section 3(b) shall be informed of the hazards from phosgene, symptoms of overexposure, emergency procedures, and precautions to ensure safe use and to minimize exposure. First aid procedures shall be included. This information shall be posted in the work place and kept on file, readily accessible to the worker.

A continuing educational program shall be instituted for workers whose jobs may involve occupational exposure to phosgene. This is to ensure that all such workers have current knowledge of job hazards, maintenance procedures, and clean-up methods, and that they know how to use respiratory protective equipment and protective clothing. Workers should be advised that the detection of the odor of phosgene at any time indicates the need for immediate corrective procedures or withdrawal from the area. First-line supervisors shall be thoroughly informed of these hazards and procedures and should participate in the education of workers.

In addition, members of emergency teams and employees who work in areas adjacent to phosgene systems or containers, where a potential for emergencies exists, shall participate in periodic drills, simulating emergencies appropriate to the work situation. Drills shall be held at

intervals not exceeding 6 months. Drills should cover, but should not be limited to:

Evacuation procedures.
Handling of spills and leaks, including decontamination.
Location and use of emergency firefighting equipment, and handling of phosgene and chlorinated hydrocarbon systems and/or containers in case of fire.
First aid and rescue procedures, including prearranged procedures for obtaining emergency medical care.
Location, use, and care of protective clothing and respiratory protective equipment.
Location of shut-off valves or switches.
Location, purpose, and use of safety showers and eyewash fountains.
Operating procedures including communication procedures.
Entry procedures for confined spaces.

Deficiencies noted during drills shall be included in the continuing educational program, together with the required remedial actions. Records of drills and training conducted shall be kept for one year and made available for inspection by authorized personnel as required.

Information as required shall be recorded on the US Department of Labor Form OSHA-20, "Material Safety Data Sheet," shown in Appendix IV or on a similar form approved by the Occupational Safety and Health Administration, US Department of Labor.

Section 6 - Work Practices

(a) Emergency Procedures

For all work areas in which there is a potential for emergencies, procedures specified below, as well as any other procedures appropriate for a specific operation or process, shall be formulated in advance and employees shall be instructed and drilled in their implementation.

(1) Procedures shall include prearranged plans for:

(A) immediate evacuation of overexposed workers;

(B) transportation of overexposed workers;

(C) any necessary calls for assistance, including alerting medical facilities of the impending arrival of overexposed workers, and calls to suppliers or manufacturers of phosgene for any necessary technical advice;

(D) designation of medical receiving facilities and names of physicians trained in phosgene emergency procedures;

(E) reentry for maintenance or clean-up purposes of areas where phosgene leaks or spills have occurred.

(2) Approved eye, skin, and respiratory protection as specified in Section 4 shall be used by personnel essential to emergency operations.

(3) Nonessential employees shall be evacuated from hazardous areas during emergencies. Perimeters of these areas shall be delineated, posted, and secured. The employees in adjacent areas shall be trained in evacuation procedures in the event that their work areas become involved.

(4) Personnel who cannot be evacuated shall keep upwind of phosgene spills or leaks, if possible. Only personnel trained in the emergency procedures and protected against the attendant hazards shall shut off sources of phosgene, clean up spills, and control and repair leaks.

(5) In case of fire, phosgene containers shall be removed to a safe place, or cooled with water if leaks do not exist. Efforts shall be made to prevent phosgene cylinders from reaching temperatures above 50 C (122 F).

(6) Water shall not be used on phosgene leaks because accelerated corrosion of metal by phosgene in the presence of moisture will quickly make the leak worse.

(7) If possible, phosgene emissions shall be directed to an alkali scrubber or to process (ie, routed by means of appropriate valving within a closed system to a secondary holding vessel or neutralization system).

(8) Containers leaking liquid phosgene should be positioned so that gaseous phosgene is discharged through the leak until control is effected.

(9) If local emergency teams cannot cope with the emergency, assistance shall be requested from the supplier or the nearest phosgene-manufacturing facility. Telephone numbers of emergency help shall be prominently posted.

(10) Phosgene in contact with skin or eyes must be removed by immediate flushing with copious quantities of water, and immediate medical attention must be obtained. Contaminated clothing must be removed immediately. If the worker has inhaled phosgene, remove him to an uncontaminated atmosphere, give artificial respiration if required, and get immediate medical attention in accordance with Section 6(a)(1). Do not allow the victim to walk or exercise in any manner. Keep the victim quiet and warm.

(b) Control of Airborne Phosgene

Engineering controls shall be used to maintain phosgene concentrations at or below the prescribed limits. The use of completely enclosed processes is the preferred method of control for phosgene. Local

exhaust ventilation may also be effective, used alone or in combination with process enclosure. Ventilation systems shall be designed to prevent the accumulation or recirculation of phosgene in the workroom, to maintain phosgene concentrations within the limits of the recommended standard, and to remove phosgene from the breathing zones of workmen. Exhaust ventilation systems shall discharge to the outside air through a sorption or a decomposition system (eg, scrubbers containing an alkaline scrubbing medium, such as 5% sodium hydroxide solution). Ventilation systems shall be subject to regular preventive maintenance and cleaning to ensure effectiveness, which shall be verified by periodic air-flow measurements. Tempered makeup air shall be provided to workrooms in which exhaust ventilation is operating.

(c) Storage

(1) Phosgene shall be stored in unoccupied, adequately ventilated, cool, and dry rooms, or outdoors shielded from the direct rays of the sun and protected from moisture.

(2) Phosgene storage rooms shall be provided with an inspection window to permit viewing of the interior without entry.

(3) Phosgene storage areas shall be completely isolated from work areas. If separated from a work area by a common wall, all holes, ducts, doors, and passthroughs which could allow phosgene to enter other parts of the plant shall be secured and sealed. Central cooling and heating ducts shall not extend to phosgene storage enclosures.

(4) Ventilation switches and emergency respiratory protection shall be located outside storage areas in readily accessible locations which will be free of phosgene in an emergency. Fan switches

shall be equipped with indicator lights.

(5) Phosgene containers shall be secured to prevent falling, upsetting, or rolling, and shall be protected from mechanical damage, heat, moisture, and corrosion.

(6) Containers of phosgene should be used on a first-in, first-out (FIFO) basis. Storage of phosgene shall be limited to the minimum necessary for the operation.

(7) Used containers should not be stored with full containers. Full containers shall be so marked, and containers in use shall be plainly marked "In Use" to differentiate from those not in use.

(8) Other materials should not be stored with phosgene.

(9) Phosgene containers shall be frequently inspected for leaks and deterioration. If the hydrostatic test date stamped on cylinders is older than 5 years, the cylinder shall be returned to the vendor, or arrangements shall be made to have the necessary test performed.

(d) Handling and General Work Practices

(1) Written operating instructions and emergency medical procedures shall be formulated and posted where phosgene is handled or used.

(2) Prompt medical attention shall be obtained if there is known or suspected overexposure to phosgene, whether or not symptoms are present.

(3) Returnable phosgene containers shall not be washed out with water.

(4) Safety valves and vents for phosgene equipment shall discharge through absorbers or neutralizers (decomposition system).

(5) Phosgene containers and systems shall be inspected daily for leaks. All phosgene equipment including valves, fittings, and connections shall be checked for tightness and good working order. All newly made connections shall be checked for leaks immediately after phosgene is admitted. Needed repairs and adjustments shall be made promptly.

(6) Appropriate precautions shall be taken to keep phosgene and phosgene equipment free of moisture. Piping, valves, and containers shall be capped or closed when not in use to keep atmospheric moisture out of the system.

(7) Transportation and use of phosgene shall comply with all applicable federal, state, and local regulations.

(8) When phosgene containers are being moved, or when they are not in use and are disconnected, valve protection covers shall be in place. Containers shall be moved only with the proper equipment and shall be secured to prevent dropping or loss of control while moving. Slings or magnetic devices shall not be modified, altered, or repaired except as normally intended by the supplier.

(9) Valves and pumps shall be readily accessible and should not be located in pits and congested areas.

(10) Discharge rates of containers of phosgene may be increased by use of warm air or warm water. Steam, boiling water, or direct flame shall not be used. Cylinder temperatures shall not exceed 50 C (122 F).

(11) Containers discharging liquid phosgene shall not be connected to manifolds. Phosgene delivery tubes and pipes from other than

high-pressure containers should not be immersed in other liquids without interposing a check valve or a trap to prevent back siphonage.

(12) The amount of phosgene used from a container shall be determined by a positive method (eg, weighing the preweighed container).

(13) New gaskets shall be used each time phosgene system connections are made.

(14) Welding or burning on tanks or equipment which have contained phosgene shall take place only after such tanks or equipment have been thoroughly purged with a dry inert gas, vented to a sorption or decomposition system. Steam or water shall not be introduced to the tanks, system, or equipment. Phosgene equipment, containers, or piping shall not be repaired while in service.

(15) Before phosgene is admitted to a system, the system shall be thoroughly cleaned, dried, and tested, using previously formulated procedures.

(16) Personnel shall not work alone when phosgene is first admitted to a system or while repairing leaks.

(17) Containers and systems shall be handled and opened with care. Approved eye, skin, and respiratory protection shall be worn while opening, connecting, and disconnecting phosgene containers and systems. When opening containers or systems, adequate ventilation shall be available to prevent inadvertent exposure to phosgene.

(18) Any odor of phosgene shall be reported to a responsible authority or an alarm sounded as soon as possible after the area has been vacated.

(e) Work Areas

(1) Where phosgene is stored, piped, handled, or used, eyewash fountains and safety showers shall be located immediately outside the area. They shall be readily accessible and shall be inspected frequently and kept in good working order.

(2) Enclosed phosgene work areas shall be equipped with at least 2 exits, remote from each other, to allow escape into uncontaminated areas in case of emergency. Doors shall open outward.

(3) Unauthorized personnel shall be prohibited from entering areas where phosgene is handled or used.

(4) Wherever possible, phosgene installations shall be outdoors. If it is necessary that such installations be indoors, workers should operate from a pressurized control room supplied with fresh air from an area remote from any possible source of phosgene contamination.

(5) For reentry purposes, at least 2 sets of self-contained breathing apparatus as specified in Table I-1 shall be located outside each work area where phosgene is handled, used, stored, or formed. In case of emergency, they shall be accessible without entry into contaminated areas. Employees shall be trained and drilled in their use.

(6) Phosgene shut-off valves shall be conspicuously marked and employees shall be familiarized with their use. Access to shut-off valves shall be unobstructed. Work areas shall be kept clean and orderly.

(f) Waste Disposal

(1) Disposal of waste phosgene shall conform to all applicable local, state, and federal regulations.

(2) Phosgene shall not be allowed to enter drains or sewers.

(3) Appreciable discharges of phosgene shall be passed through an adequate decomposition system, such as a scrubbing tower utilizing sodium hydroxide or ammonium hydroxide, or through a sorbent system.

(4) Solid sorbents should be chosen so that desorption of phosgene is unlikely. Heating of the solid sorbent should be avoided.

(g) Confined Spaces

(1) Entry into confined spaces such as tanks, pits, tank cars, barges, process vessels, and tunnels shall be controlled by a permit system. Permits shall be signed by an authorized employer representative certifying that preparation of the confined space, precautionary measures, and personal protective equipment are adequate, and that precautions have been taken to ensure that prescribed procedures will be followed.

(2) Confined spaces which have contained phosgene shall be inspected and tested for oxygen deficiency, phosgene, and other contaminants and shall be thoroughly ventilated, cleaned, neutralized, and washed, as necessary, prior to entry.

(3) Inadvertent entry of phosgene into the confined space while work is in progress shall be prevented by disconnecting and blanking of phosgene supply lines.

(4) Confined spaces shall be ventilated while work is in progress to keep the concentration of any phosgene present below the standard and to prevent oxygen deficiency.

(5) Individuals entering confined spaces where they may be exposed to phosgene shall be equipped with adequate respirators and suitable harnesses with lifelines tended by another worker outside the

space who shall also be equipped with the necessary protective equipment.

(h) Enclosed Spaces

Enclosed spaces (rooms, buildings, etc) which ordinarily are safe to enter but which, due to the failure of a system inside, could contain hazardous concentrations of phosgene should have a continuous automatic monitor (see Appendix III) set to sound an alarm which is audible inside and outside the enclosed space if phosgene concentrations exceed the ceiling concentration limit. A warning light is recommended as a substitute for a bell in noisy areas. If such areas are not monitored in this way, they shall be entered only if the worker is under observation by a coworker and if the worker has in his possession a respirator suitable for escape.

(i) Miscellaneous

Unless the potential for inadvertent phosgene generation is anticipated and engineering controls are implemented, chlorinated hydrocarbons shall not be exposed to high temperatures or ultraviolet radiation.

Section 7 - Sanitation Practices

(a) Plant sanitation shall meet the requirements of 29 CFR 1910.141.

(b) Escape routes near phosgene control equipment shall be kept clear, reflecting general good housekeeping practices.

(c) Appropriate locker rooms shall be available for changing into required protective clothing in accordance with 29 CFR 1910.141(e). Clothing contaminated with liquid phosgene shall be immediately removed and

placed in a closed container in a well-ventilated area for later disposal or decontamination.

(d) Food should not be stored, prepared, dispensed, or eaten in phosgene work areas.

Section 8 - Monitoring and Recordkeeping Requirements

(a) Workroom areas where it has been determined, on the basis of an industrial hygiene survey or the judgment of a compliance officer, that environmental levels of phosgene are less than half of the TWA limit should not be considered to have phosgene exposure. Records of these surveys, including the basis for concluding that air levels of phosgene are below half of the TWA limit, shall be maintained until a new survey is conducted.

(b) Area Monitoring

Continuous automatic monitoring is recommended in any work area where an initial industrial hygiene survey indicates that a potential phosgene exposure exists. Such monitoring devices (see Appendix III) should have an audible or a visible alarm (light) which is triggered whenever the ceiling concentration limit is exceeded.

(c) Personal Monitoring

(1) Initial Monitoring

Within 6 months of the promulgation of this standard, each employer who has a place of employment in which phosgene is stored, handled, used, or formed shall design and implement a monitoring program which shall identify and measure or permit calculation of the exposure of all employees exposed to phosgene.

(2) Normal Monitoring

(A) Routine monitoring of employee exposure shall be conducted at 3-month intervals unless otherwise indicated by a professional industrial hygienist and whenever introduction of a production, process, or control change indicates a need for reevaluation.

(B) If an employee monitoring program measurement reveals that an employee is exposed in excess of the recommended environmental limits, the exposure of that employee shall be measured at least once every 2 weeks, control measures required by Section 6(b) shall be implemented, and the employee shall be notified. When two consecutive biweekly determinations reveal that employee exposure no longer exceeds either of the recommended environmental limits, routine monitoring may be resumed.

(3) Exposure Measurement

In all personal monitoring, samples representative of the exposure in the breathing zone of the employee shall be collected. Procedures for sampling, calibration of equipment, and analysis of phosgene samples shall be as provided in Appendices I and II, or by any method shown to be equivalent in precision, accuracy, and sensitivity to the methods specified.

An adequate number of samples shall be collected to permit construction of a TWA and peak exposure value for every operation or process. Variations in work and production schedules shall be considered in deciding when samples are to be collected. The minimum number of representative TWA determinations for an operation or process shall be based on the number of workers exposed as provided in Table I-2, or as otherwise indicated by a professional industrial hygienist.

TABLE I-2

SAMPLING SCHEDULE

Number of Employees Exposed	Number of TWA Determinations
1 - 20	50% of the total number of workers
21 - 100	10 plus 25% of the excess over 20 workers
Over 100	30 plus 5% of the excess over 100 workers

(d) Recordkeeping

Employers shall maintain records of any accidental phosgene release requiring evacuation, and results of all exposure measurements, environmental surveys, and medical examinations performed as required by Section 2 of this chapter. Such records shall indicate the type of personal protective devices, if any, in use at the time of sampling. Records of environmental monitoring shall be maintained and shall be available to the authorized representatives of the Secretary of Health, Education, and Welfare, and of the Secretary of Labor. Each employee shall be able to obtain information on his own environmental exposure. Such

records shall be maintained for at least 5 years after the individual's employment is terminated.

II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon which were prepared to meet the need for preventing occupational diseases arising from exposure to phosgene. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare, under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to "...develop criteria dealing with toxic materials and harmful physical agents and substances which will describe...exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience...."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultation with others, formalized a system for the development of criteria upon which standards can be established to protect the health of workers from exposure to hazardous chemical and physical agents. It should be pointed out that any criteria and recommended standard should enable management and labor to develop better engineering controls resulting in more healthful work environments. Simply complying with the recommended standard should not be the final goal.

These criteria for a standard for phosgene are part of a continuing series of criteria developed by NIOSH. The proposed standard applies to the processing, manufacture, use of, or other occupational exposure to phosgene as applicable under the Occupational Safety and Health Act of 1970. The standard was not designed for the population-at-large, and any extrapolation beyond occupational exposures is not warranted. It is intended to (1) protect against injury from phosgene, (2) be measurable by techniques that are valid, reproducible, and available to industry and

official agencies, and (3) be attainable with existing technology.

The development of the recommended standard for occupational exposure to phosgene has revealed deficiencies in the data base in the following areas:

(1) epidemiologic studies of workers exposed to phosgene for extended periods;

(2) chronic animal exposure studies at low levels of phosgene;

(3) improvement of the sensitivity of sampling and analytical methods for personal monitoring;

(4) testing of automatic, continuous monitoring systems and associated alarms.

These gaps in our knowledge of phosgene should be filled.

III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure

Phosgene is a colorless gas at normal temperatures and pressure. When liquified under pressure or refrigeration, it is a colorless-to-light yellow liquid. [1] In low concentrations, its odor has been variously described as resembling that of musty hay [2] or green corn. [3] Phosgene is easily manufactured by passing chlorine and excess carbon monoxide over activated carbon. [3] Shortages of chlorine and attendant high prices have somewhat restricted the manufacture of phosgene in recent years. [4] Some phosgene can also be produced by the decomposition of chlorinated hydrocarbons by heat or by ultraviolet radiation. [5,6,7,8,9] Relevant properties of phosgene are presented in Table XIII-1. [1,2,10,11,12]

Phosgene was first used as a chemical warfare agent during World War I. Its use in industry is a relatively recent development. [3] Accordingly, much of the literature on phosgene is concerned with its military applications. Phosgene production in the United States in 1957, the first year the US Tariff Commission started reporting phosgene output, was only 5 million pounds. [3] In 1967, production reached 350 million pounds, [3] in 1971, 530 million pounds, [13] while in 1972, it had increased to 657 million pounds. [14] In contrast to these figures, sales in 1971 were only 11,215,000 pounds [13] and 11,678,000 pounds in 1972. [14] The apparent discrepancy between production and sales is due to the fact that the major portion of the phosgene manufactured is for "captive" use (use by the phosgene manufacturer), while the relatively small remainder is sold. [3] In 1974, phosgene was being produced in 18 plants

and capacity was on the increase. [4] It has been estimated that demand will be 1,630 million pounds in 1978. [4]

The relatively recent revitalization of the phosgene industry and the rapidly increasing demand are due largely to the use of phosgene in the synthesis of isocyanates, which are starting materials for polyurethane resins. Production of isocyanates accounted for about 75% of all phosgene produced in 1967. [3] In 1974, the uses of phosgene were distributed as follows: production of toluene diisocyanate, 62%; other polymeric isocyanates, 23%; polycarbonates, 6%; pesticides, carbonates, and "specialties," 9%. [4]

Some occupations with potential exposure to phosgene are listed in Table XIII-2. [15] NIOSH estimates that 10,000 workers have potential occupational exposure to phosgene during its manufacture and use.

Historical Reports

Berghoff [16] reported his observations of 2,000 cases of exposure to war gases during World War I. These included chlorine, mustard gas, and phosgene. The main complaint expressed by those exposed to phosgene was a weakness which developed as early as 2 hours or as late as 3 days after exposure. This weakness lasted for weeks or months. The author also noted that emphysematous patients had a more protracted convalescence than those classified as bronchitic.

Effects on Humans

Wells et al [17] published a detailed report on effects of barely detectable concentrations of phosgene on humans. Fifty-six military personnel, without upper respiratory problems, were exposed to increasing concentrations of phosgene until all the subjects could detect phosgene by odor. The authors reported that 50% of "technically trained" (without further clarification) observers detected phosgene at a concentration of 6.1 mg/cu m (1.5 ppm). Thirty-nine percent detected it at a concentration of 4.7 mg/cu m (1.2 ppm). None detected it below a concentration of 1.5 mg/cu m (0.4 ppm). The authors exposed the subjects to phosgene in increasing concentrations until they detected an odor. No effort was made to distinguish phosgene from other odors. No effects other than odor detection were reported.

Leonardos et al [18] studied phosgene odor thresholds using a panel of 4 members. The odor threshold was defined as the first concentration at which all 4 members recognized the odor. They distinguished this from the "detection threshold" which they felt was neither reliable nor reproducible. They determined a "hay-like" odor threshold for phosgene of 1.0 ppm.

Thiess and Goldmann [19] described their experience with 109 cases of accidental phosgene inhalation, including one fatality, in one industrial plant. The patients reported that they were exposed to only 1 or 2 whiffs of phosgene each, but some exposures were probably more severe. No further quantitation was described by the authors. Of these cases, 70 had insignificant clinical problems, hence were not studied in detail by the industrial physician. No details were given concerning these so-called

insignificant complaints. Of 31 cases in which X-ray studies were performed after exposure, 5 showed pulmonary abnormalities upon radiological examination. Only 3 of these showed the characteristic picture of pulmonary edema. The authors reported that the patients followed the "typical symptoms and course of phosgene poisoning: after an almost unnoticed inhalation,...a certain symptom-free latent period of 2 to 8 hours follows, and then the typical pulmonary edema (occurs)...." These three cases were described in detail.

A 19-year-old chemical laboratory assistant was accidentally sprayed with an unknown volume of liquid phosgene. The gas mask he was wearing was not leakproof, hence phosgene penetrated the mask. The mask and upper clothing were removed immediately. No first aid was administered. Upon admission to the hospital a little more than half an hour after exposure, the patient was observed to be in respiratory distress. Chest films showed infiltration of the lungs and pulmonary edema. Therapy consisted of phlebotomy, digitalization, intravenous fluids, and antibiotics. Three weeks of hospitalization were required before the patient could return home. No sequelae were described.

A 20-year-old chemical laboratory assistant was sprayed in the face with chlorobenzene saturated with phosgene under pressure. It was estimated that one mole of gaseous phosgene was released during the accident. First aid consisted only of washing the face and hair in water. Five hours after exposure, the patient felt a slight pressure on his chest. Eight hours after exposure, he became dyspneic and expectorated bloody sputum. He was admitted to the hospital one hour later. Chest films showed pulmonary edema. Thirteen days of hospitalization, which included

treatment with corticosteroids, digitalis, and oxygen, were required to resolve his pulmonary problems.

The fatal accident involved a 55-year-old mason who was presumed to have been exposed to phosgene released by chipping of brick which had possibly adsorbed phosgene. In this case, phosgene was a byproduct in the production of aluminum chloride. An analysis of the apparatus the patient worked on revealed that 2.5 liters of interstitial air volume in the brick at 360-400 C was available for absorbing the phosgene. The amount of dust inhaled by the patient is unknown. He was exposed for 30 minutes and first complained of dyspnea about 2 hours after completing the job. No first aid was given. Five hours after exposure, he was admitted to the hospital in severe respiratory distress. Chest films showed pulmonary edema. Despite phlebotomy and treatment with digitalis and diuretics, the patient died of acute right heart failure about 14 hours after his initial exposure.

Two cases of phosgene exposure were reported by Gerritsen and Buschmann. [6] They were due to accidental formation of phosgene from chlorinated hydrocarbons. Both cases involved the use of chemical paint removers in poorly ventilated areas heated by portable kerosene stoves. The first case involved a 52-year-old man who was exposed for an unknown period. He noted respiratory irritation soon after beginning work but persisted working for several hours. Chest symptoms occurred thereafter and the patient, upon examination, exhibited signs of pulmonary congestion. Approximately 5 hours later, the patient went into frank pulmonary edema and died within a few hours. Autopsy showed extensive degenerative changes in the epithelium of the trachea, bronchi, and bronchioli, together with hemorrhagic edematous focal pneumonia.

The second case [6] involved a 38-year-old woman, in her 7th month of pregnancy, who was exposed in a similar manner for 3 hours in the afternoon. That evening hemoptysis occurred. The next morning, symptoms worsened and she was hospitalized. A chest film upon admission showed pulmonary edema. After 8 days' hospitalization, she was released even though her chest film did not yet show a complete return to normal. After 2 months, she gave birth to a healthy child. This is the only case found which reported phosgene exposure during pregnancy.

In attempting to reproduce the circumstances of exposure of the above 2 cases, [6] it was found that methylene chloride was rapidly decomposed with phosgene being the main decomposition product when methylene chloride was exposed to heat in a poorly ventilated area. The authors stated that this was in contrast to the results reported by Little [20] when methylene chloride was decomposed by hot surfaces and low amounts of phosgene were produced in comparison with hydrogen chloride and chlorine.

Another case of possible phosgene poisoning resulting in death was reported by Spolyar et al. [5] The case involved a chlorinated solvent degreaser which was inadvertently filled with trichloroethylene instead of perchloroethylene. The operator of the degreaser was found dead 3 1/2 hours after exposure began and 1 hour after he reported that fumes were escaping from the apparatus. Autopsy showed pulmonary edema consistent with exposure to phosgene. It was assumed that the trichloroethylene vaporized and passed through the firebox of a nearby space heater, with decomposition of the trichloroethylene and the production of phosgene. Cause of death was consistent with phosgene exposure but it was suggested that trichloroethylene might have contributed to the circulatory collapse.

An attempt was made to reproduce the environmental situation. [5] Sampling of the breathing zone of the operator revealed a phosgene concentration of 15 ppm. The analytical method was altered to correct for interferences by trichloroethylene at 3,300 ppm in air, the estimated trichloroethylene concentration at the time of exposure. During the test simulation, trichloroethylene levels exceeded 10,000 ppm after 1 hour and 20 minutes of degreaser operation.

Glass et al [21] reported a case of poisoning attributed to phosgene following the welding of a metal which was damp with trichloroethylene used for cleaning purposes. After 4 1/2 hours' exposure, the worker noted respiratory symptoms and felt unwell. He returned home, but the next morning he was dyspneic. Chest films taken 24 hours after exposure and 90 days later showed the diaphragm below the eleventh rib posteriorly with limited excursion and clear lung fields. Pulmonary function tests, including spirometry, carbon monoxide uptake, and arterial blood gases, abnormal at first, improved over a 3-month period following exposure. The authors' impression was that the patient suffered from chronic bronchitis which was exacerbated by phosgene. Unfortunately, no studies of phosgene in the air were conducted in the workplace under conditions simulating that of the original exposure.

Derrick and Johnson [22] reported a case of presumed phosgene exposure due to the breakdown of trichloroethylene by cigarette smoking. The patient had worked as a drycleaner for 3 months. Studies indicated that the average concentration of trichloroethylene in the room was 488 ppm. The authors indicated that this level would be exceeded when clothing was removed from the cleaning machine. The patient was known to smoke 40

cigarettes/day. He frequently smoked in the cleaning room. He left work at 4:00 p.m., and about 90 minutes later he collapsed and died. An autopsy showed pulmonary edema. Phosgene was believed to have been generated by the decomposition of trichloroethylene in contact with the hot tip of a burning cigarette. This theory is contradicted by the work of Little [20] who measured phosgene in the effluent gas of cigarettes and did not detect any in atmospheres containing trichloroethylene, chloroform, carbon tetrachloride, perchloroethylene, or even small amounts of phosgene.

Everett and Overholt [23] reported a case of phosgene poisoning but gave no details of exposure other than "massive exposure to phosgene." Initial symptoms were burning of the eyes and coughing. These cleared after a few minutes, but dyspnea occurred in 3 hours. X-ray studies showed pulmonary edema which resolved over 7 days of hospitalization and treatment which included antibiotics, corticosteroids, and oxygen. The patient remained well during the ensuing 2 years.

The Bureau of Engineering Safety, Department of Labor and Industry, State of New Jersey, [24] reported one fatality among 6 employees exposed to phosgene at unknown concentrations in separate accidents over a 2-year period. The exposures occurred in a plant conducting "phosgenation" where measurements of air concentrations were normally reported to be below 0.1 ppm. Subsequently, all phosgene operations were stopped because of inadequate engineering controls.

Delepine [25] described 2 cases of fatal phosgene poisoning. The first man had his clothing saturated with phosgene and was treated almost immediately. He appeared well but experienced symptoms 6 hours after exposure. Treatment (details not given) was temporarily helpful, but the

patient died 11 hours later. The second man was exposed as a result of the explosion of a phosgene cylinder. Death occurred 22 hours after exposure. At autopsy, both cases showed evidence of severe irritation of the respiratory tract with almost complete shedding of the laryngeal, tracheal, and bronchial epithelium.

English [8] reported a case of poisoning attributed to phosgene in a 67-year-old male with several years' history of chronic bronchitis and a quiescent duodenal ulcer. After an 8-hour exposure in a room heated by a stove burning paraffin in which paint-strippers containing chlorinated hydrocarbons had been used, the worker experienced dyspnea. The next morning, his symptoms increased and he was hospitalized. Chest X-rays showed diffuse bronchiolitis. Despite treatment, dyspnea persisted for 4 days in the hospital. He was discharged after 6 weeks. English stated that phosgene dissolved in saliva irritated the alimentary mucosa and, hence, was responsible for reactivation of a duodenal ulcer in this patient. He cited no authorities for this statement or clinical or experimental evidence to support it. No other references to the effects of phosgene on gastrointestinal mucosa were found other than Cherkes' [26] statement that stasis and venous hyperemia occur in the gastrointestinal tract as a result of pulmonary edema.

Seidelin [7] reported a case of probable phosgene inhalation leading to pulmonary edema in a 16-year-old woman. This occurred after she had used a carbon tetrachloride fire extinguisher in an enclosed space. Inhalation of smoke and fumes resulted in immediate coughing. Six hours later, she developed respiratory symptoms and subsequently was admitted to the hospital with pulmonary edema. Complications ensued including

mediastinal emphysema and bilateral pneumothoraces. Oxygen therapy resulted in considerable clinical improvement in 8 days, but she was unable to leave the hospital until 13 days after exposure.

Stavrakis [27] described 7 cases of phosgene exposure. The first was a worker who developed dyspnea, cough, and chest pain 4 hours after exposure, which were severe enough to bring him to a hospital emergency room. Treatment with hexamethylenetetramine was given immediately, followed by standard therapy consisting of steroids, oxygen, and antibiotics. He was discharged in good health after 5 days. The other 6 workers were exposed when a pipe ruptured and released phosgene. The extent of exposure was not described. One heavily exposed worker was treated immediately with hexamethylenetetramine. He remained asymptomatic until his discharge 24 hours later. Another worker, similarly exposed, waited until symptoms occurred before seeking treatment. He died despite treatment with hexamethylenetetramine. Four others, who were treated in the symptomatic stage, required hospitalization for various periods until recovery occurred.

In 1946, Galdston et al [28] reported studies of 6 cases of acute exposure to phosgene with residual effects up to 19 months after the last known acute exposure. Evaluation of each patient included physical examinations, chest X-rays, pulmonary function tests, and a psychiatric summary. These cases shared a common background of brief single exposures to phosgene at unknown concentrations which usually led to delayed pulmonary edema. One of the cases is interesting in that exposure occurred at a hood which contained an ampule of only 40 ml phosgene. All were treated at Johns Hopkins Hospital and released, some returning to a normal

work routine. However, follow-up examinations revealed that all had lingering complaints, and although physical examinations and chest X-rays up to 19 months later were generally normal, pulmonary function tests always revealed some abnormalities consistent with beginning pulmonary emphysema. The authors felt that psychological factors contributed to the lingering symptomatology. Their findings are summarized in Table III-1.

Cherkes, [26] in an extensive review of the literature concerning the clinical course of acute phosgene exposure, noted that most fatalities occur during the first 24-48 hours. He reported that most patients dying within the first 72 hours died of pulmonary edema or cardiac problems. Those dying later usually succumbed as a result of complications, such as infection (usually pulmonary), thrombosis, or embolism. He gave no source for these statements other than "according to the data of various authors." The clinical course following phosgene exposure reported by Cherkes is generally in agreement with other reports following human and animal exposure. [5,6,21,25,27,29,30,31]

Ardran [32] pointed out that many victims of phosgene poisoning showed radiological evidence of increased lung volumes. His experiments with dogs [33] indicated that animals that failed to develop an increase in lung volume after phosgene exposure also failed to develop pulmonary edema. This test had been used by him clinically. [32] He reported that, if an expiratory lung film shows evidence of an increase in volume after exposure to phosgene, then pulmonary edema may be expected. He stated that he had looked for this sign in humans exposed to lung irritants and that never, in 20 years, had he found pulmonary edema to develop in the absence of antecedent increased lung volume. There has been no independent

TABLE III-1

SUMMARY OF CLINICAL OBSERVATIONS AND DATA ON STUDIES PERFORMED AFTER ACUTE EXPOSURES

Case Number*	1	2	3	4	5	6
Age	38	39	30	48	43	49
Months after accident**	14	6	6	3	5	5
Months worked with phosgene	6	12	18	24	2	1
Chronic symptoms	A	N	N	A	A	A
Physical signs						
Acute	A	A	A	A	N	N
Chronic	N	N	N	B	N	N
Roentgenogram of chest	N	N	N	N	N	N
Volume						
(Vital capacity + % residual air) = Total capacity	N	N	N	A	N	N
	B	N	N	B	N	N
Intrapulmonary mixing of gases	N	N	B	A	N	B
Pulmonary emptying	N	N	N	A	N	B
Resting pattern of breathing						
High rate	N	A	A	A	A	A
Low tidal air	N	N	A	B	A	A
High min. volume	N	A	A	A	A	A
Low oxygen extraction	B	A	A	B	A	A
Exercise pattern of breathing						
High rate	N	B	B	B	A	A
Low tidal air	B	N	B	B	N	A
Low oxygen extraction	N	N	N	B	A	A
Arterial blood						
At rest	N	A	N	N	A	N
After exercise	N	A	N	N	N	-
After oxygen administration	-	N	-	N	A	-
Breath holding	N	N	N	A	A	N
Voluntary breathing capacity	N	A	A	N	A	N
Postural tests	N	N	N	N	N	N
Cardiac output	N	A	N	N	-	N

A = Definitely abnormal B = Borderline abnormal
N = Normal - = Not done

* Listed in order of severity of exposure.

** Applies to all special studies except arterial blood and alveolar air oxygen and carbon dioxide tensions and cardiac output which were performed 4-8 months later. Symptoms, physical and X-ray findings were unchanged on reexamination of all available patients (except No. 5) 4-8 months later.

From reference 28

confirmation of his interesting findings, and he gave no pulmonary function test data on his patients.

Steel [34] described 2 cases of exposure to phosgene at low concentrations (figures for duration or concentration not stated). Both patients developed delirium, fever, tachycardia, tachypnea, and a painful cough. The more exposed patient developed pulmonary edema; the other showed only acute bronchitis. Steel noted that both patients developed amnesia about their exposure. He stated that he regarded neither fever nor amnesia as characteristic of exposure to phosgene.

One of the few publications relating to workers with multiple exposures to phosgene at low concentrations over prolonged periods is that of Galdston et al. [35] Their observations are summarized in Table III-2. The study involved the pulmonary function, cardiovascular and psychiatric status of 5 workers who had repeated exposures to small amounts of phosgene during the course of 18-42 months. In none of the cases was notation made of odor detection by the patients during exposure, prior to the development of more serious symptoms. The first patient, age 32, had a noncontributory past history except for conjunctivitis and laryngitis after working with mustard gas during 2 time periods. Several exposures to phosgene caused a feeling of chest constriction, dizziness, headaches, blurred vision, and mental confusion. The same year, he experienced severe irritation of the throat from inhaling chlorine. He worked with phosgene for another 4 months and noted chest tightness, dyspnea on exertion, and muscular twitching he ascribed to recent minor exposures to phosgene. The findings of his physical examination and chest roentgenogram were normal; however, pulmonary function studies showed a decrease in vital capacity, impaired

intrapulmonary gas mixing and other changes consistent with pulmonary emphysema.

The second patient, [35] age 50, also had a noncontributory past history prior to working with phosgene. He had had numerous minor exposures to phosgene which were usually followed by a sense of constriction in the throat, dyspnea, cough, nausea, and vomiting. After working with phosgene, he had a productive cough which occasionally tasted of phosgene. Findings from his physical examination were normal, but his chest roentgenogram and pulmonary function studies were consistent with pulmonary emphysema.

The third patient, [35] age 24, had had a history of asthma since childhood. He had several minor exposures to chlorine before working with phosgene. On 6 occasions, he inhaled enough phosgene to induce coughing, choking sensations, nausea and vomiting, headache, and sweating, which disappeared the day following exposure. Physical examination demonstrated only thoracic kyphosis and bilateral basilar rales. Roentgenograms and pulmonary function studies were consistent with pulmonary emphysema.

The fourth patient, [35] age 31, had chronic tonsillitis, otitis, and adenoiditis apparently prior to his phosgene exposure. He also had minor symptomatic episodes of exposure to chlorine and mustard gas. After about 6-9 months of exposure to phosgene, physical examination showed a perforated right eardrum and bilateral basilar rales. Roentgenograms of the lungs showed what was described as an old obliteration of the left costophrenic angle. Pulmonary function studies were consistent with pulmonary emphysema.

The fifth patient, [35] age 26, had worked with both phosgene and chlorine during separate periods. He had a few minor exposures to phosgene which resulted in conjunctival irritation, dyspnea, and headache. Physical examination and roentgenographic studies were normal. Pulmonary function studies showed only a reduction in voluntary breathing capacity. The authors concluded that "emphysema of the lungs may develop after chronic exposure to phosgene."

This study is an important one in that it deals directly with the problem of repeated minor exposures to phosgene. Unfortunately, Galdston et al [35] did not comment on how these patients were selected or on any quantitation of the phosgene exposures. This paper also did not consider continuous exposures at a low level of phosgene during a full workday and workweek over an extended period. It is, however, the only paper available with clinical and laboratory data collected on humans with repeated exposures to phosgene. (see Table III-2)

TABLE III-2

SUMMARY OF CLINICAL OBSERVATIONS AND DATA ON STUDIES PERFORMED AFTER CHRONIC EXPOSURES

Case Number*	1	2	3	4	5
Age	32	50	24	31	26
Months worked with phosgene	42	36	30	16	30
Chronic symptoms	A	A	A	A	A
Physical signs					
Acute	N	N	N	N	N
Chronic	N	B	A	N	N
Roentgenogram of chest	N	A	A	N	N
Volume					
(Vital capacity + % residual air) = Total capacity	A	N	B	N	N
	A	B	A	B	N
Intrapulmonary mixing of gases	A	A	A	A	N
Pulmonary emptying	N	B	A	A	N
Resting pattern of breathing					
High rate	N	A	A	A	N
Low tidal air	N	A	A	B	N
High min. volume	N	A	A	A	N
Low oxygen extraction	N	N	B	A	N
Exercise pattern of breathing					
High rate	N	B	B	B	N
Low tidal air	N	B	B	B	N
Low oxygen extraction	N	N	A	A	N
Arterial blood gases**					
At rest	N	A	N	N	-
After exercise	N	N	A	A	-
After oxygen administration	N	-	-	-	-
Breath holding	N	A	-	N	N
Voluntary breathing capacity	N	A	A	N	A
Postural tests	N	-	-	-	-
Cardiac output	N	A	N	N	-

A = Definitely abnormal
B = Borderline abnormal
N = Normal
- = Not done

* Listed in order studied

** Arterial blood oxygen, alveolar air oxygen and carbon dioxide tension studies at rest and after exercise were performed 4-8 months after all other studies were completed. Symptoms, physical and roentgenographic findings were unchanged on reexamination of all available patients (all but one) at that time.

From reference 35

Epidemiologic Studies

Levina et al [36] described the working environment in the monuron, 3-(p-chlorophenyl)-1,1-dimethylurea, industry. Phosgene is involved in its synthesis and was found to be contaminating 90 workers' production areas at a concentration of 1.0-2.0 mg/cu m (0.25-0.5 ppm) over a 6-month period under investigation. According to Smelyanskiy and Ulanova, [37] the permissible level for the USSR was 0.5 mg/cu m (0.125 ppm). Other contaminants included chlorobenzene, dimethylamine, and parachlorophenylisocyanate. Levina et al [36] reported no pulmonary problems in these workers, but did not describe searching for them.

Levina and Kurando [38] reported their studies of a plant manufacturing a weed killer (isopropylphenylcarbamate) using phosgene, isopropyl alcohol, aniline, and caustic soda as raw materials. Although a closed process was used, phosgene was found in 30% of all air samples, most frequently at a concentration of 0.5 mg/cu m (0.125 ppm). A total of 89 workers were studied for evidence of hematological abnormalities. Methemoglobinemia and anemia were detected which were attributed to the weed killer and aniline. No mention was made of pulmonary problems.

At a plant where phosgene is manufactured, the medical records of all exposed workers (326) were compared with those of 6,288 nonexposed workers. (AF Myers, written communication, November 1974) Pulmonary function, lung problems, and deaths related to lung problems were tabulated for both groups. The data were taken to indicate that there were no chronic lung problems related to working in these phosgene operations. By using the age distribution of employees and pensioners and comparing their deaths from lung problems with those expected from a similar age group (described as

taken from National Statistics) not exposed to phosgene, no increase in lung-related deaths was noted in the phosgene-industry workers. The details of pulmonary function testing were not provided. The results of a limited program of air sampling conducted during a 2-month period were provided. Fifteen personal air samples collected for 20-minute periods and analyzed using the NBP method used by AF Myers (written communication, November 1975) and described in Chapter IV of this document showed concentrations ranging from nondetected to 0.08 mg/cu m with an average concentration of 0.012 mg/cu m. From a total of 56 fixed-position samples collected for 2-hour or 20-minute periods, 51 samples showed concentrations ranging from nondetected to 0.52 mg/cu m (ND-0.13 ppm). The remaining 5 samples showed "off-scale" measurements (greater than 0.55 mg/cu m) reportedly due to leaks.

Animal Toxicity

Clay and Rossing [39] exposed 25 mongrel dogs to phosgene at a concentration between 24 and 40 ppm for 30-minute periods at a rate of 1-3 exposures/week. Those exposed once or twice showed acute bronchiolitis and peribronchiolitis involving terminal and respiratory bronchioles. The trachea and bronchi were visually unaffected and the proximal bronchioles were seldom damaged. Those exposed 4-10 times had chronic bronchiolitis of the proximal and intermediate portions of the respiratory bronchioles. The animals exposed 30-40 times showed changes which were described as resembling those of early emphysema.

Box and Cullumbine [40] studied the problem of an apparent reduction in susceptibility to phosgene intoxication by prior exposure. They exposed

rats for 10 minutes to phosgene at concentrations of 80 mg/cu m (20 ppm). Five days later, the preexposed animals and an equal number of control animals were exposed to phosgene at lethal concentrations (230-440 mg/cu m, 55-110 ppm) for 10 minutes. The mortality rate for controls was 74%, while for pregassed animals it was only 33%. They attributed this finding to rapid and shallow breathing caused by pulmonary damage in the first exposure.

Rinehart and Hatch, [41] using low concentrations of phosgene (0.5-4 ppm for 5-480 minutes) on rats, attempted to work out the validity of the concentration-time product (Ct) in ppm-minutes as a measure of dose of sublethal exposures to phosgene. On the basis of the responses (expressed in terms of impaired pulmonary gas exchange capacity as measured by the decreased rates of uptake of carbon monoxide and ether) of 118 Wistar rats, the authors concluded that the Ct was a suitable way to express the magnitude of the dose, and that low-level exposure to phosgene with a Ct equal to or less than 100 ppm-minutes caused increased resistance to breathing and poorer distribution of air within the lungs. Above a Ct of 100 ppm-minutes, decrease in diffusion capacity became more important. They attributed this to differences in the major site of action, ie, the respiratory bronchioles in the first case, and the alveoli in the second. The authors noted that above a Ct of 30 ppm-minutes gas exchange capacity decreased directly with the logarithmic increase in Ct. Rinehart and Hatch [41] noted no significant effect of phosgene on the test animals' pulmonary performance when subjected to exposures less than a Ct of 30 ppm-minutes. The animals' exposures were varied to cover a Ct product range of 12-360 ppm-minutes.

Gross et al [42] studied the effect of low concentrations (0.5-4 ppm for 5-480 minutes) on rats. They found that they could produce a chronic pneumonitis which was reversible but left detectable lesions for up to 3 months. They felt they could explain this by the fact that low dosages of phosgene merely irritated the pulmonary alveolar epithelium, resulting in proliferation. The more severe exposures of phosgene usually reported in the literature destroyed the surface epithelium and attacked the underlying alveolar capillaries, thus resulting in pulmonary edema. The authors noted that the severity of the chronic pneumonitis correlated well with the Ct value of the phosgene exposure and seemed to be largely independent of the concentration of the gas in the same bracket of Ct values. Animals in the study by Gross et al [42] were subjected to Ct products ranging from 13 to 360 ppm-minutes. In the 18 rats exposed to phosgene with Ct products equal to 30 ppm-minutes or less, 5 (28%) showed no abnormalities on pathologic examination; 11 (61%) showed slight chronic pneumonitis; and 2 (11%) showed moderate chronic pneumonitis.

Wirth [43] studied the effect of low concentrations of phosgene upon cats. He reported that, if the concentration was expressed as mg/cu m, the lethal concentration x the survival time in minutes was approximately 1,000. At low concentrations (5-7 mg/cu m, 1-2 ppm), the constant was as high as 3,000. This shows that the lethal Ct product is considerably higher at low concentrations. The author felt that the increase in Ct product at low concentrations was due to detoxification and that the practical usefulness of the Ct formula was not affected by this, provided it was used within certain concentration limits.

Cordier and Cordier [44] exposed cats and guinea pigs to phosgene at concentrations of 20-25 mg/cu m (5.0-6.25 ppm) and 10-15 mg/cu m (2.5-3.75 ppm) repeatedly over several weeks. The duration of each exposure was 10 minutes. The interval between exposures was 24 hours, and the number of exposures varied from 2 to 41. Examination of the animals concentrated on body weight, organ weight, and microscopic examination of the lungs. After exposure at 20-25 mg/cu m, all animals developed pulmonary lesions, although signs of these were not detected while the animals were alive. Microscopic examination of the 15 cats exposed indicated that all but two had some degree of pulmonary edema. The remaining two showed other lung abnormalities. The degree of lung damage did not show any increase with increasing number of exposures. Therefore the authors concluded that there is no cumulative effect of phosgene at this concentration when the duration of exposure is short and the animals are given time to recover between exposures. Both cats and guinea pigs were exposed to phosgene at the lower range of concentrations of 10-15 mg/cu m. Upon microscopic examination, pulmonary edema was found in 3 of 6 cats and in none of 6 guinea pigs. Other lung changes were found, but, in general, the effect on the pulmonary alveoli was considered to be insignificant. The authors concluded that this concentration, inhaled daily for 10 minutes, seems to be the minimal concentration capable of creating edematous pulmonary zones. This minimum effect level, expressed as a Ct product (25-37.5 ppm-minutes) concurs with the minimum effect level of 30 ppm-minutes later found by Rinehart and Hatch [41] in experiments with rats using pulmonary uptake of carbon monoxide and ether to measure effect.

Koontz [45] gassed dogs with phosgene at the minimum lethal dose (undefined by the author) and then studied 95 of those that survived. One-third died or were killed by other dogs during the course of the experiment. The other two-thirds were killed at intervals from 2 to 60 weeks. About one-half of the dogs showed no or only minor lesions. Those with more significant abnormalities showed transient bronchial plugging and adjacent atelectasis. Most of the lungs took on a more normal appearance as the time from recovery increased.

Durlacher and Bunting [46] exposed 31 dogs to phosgene at concentrations averaging 0.29 mg/ liter (72.0 ppm) for 30 minutes. The animals were given a variety of treatments, including oxygen, transfusions, or venesection. The most striking findings were consolidation of one or more lobes of the lungs 4-9 days after exposure. The authors noted that "pulmonary organization occurred...and caused high mortality in spite of oxygen therapy." The oxygen therapy consisted of maintenance in an atmosphere of 60% oxygen when the arterial oxygen saturation was below 80%. No specific time for initiation of therapy other than "after exposure" was given.

Gross et al [47] described their findings concerning pulmonary reactions to toxic gases. They noted that the proliferative lesions produced by phosgene, chlorine, sulfur dioxide, nitrogen dioxide, ozone, and crotonaldehyde differed only quantitatively on a histologic basis. It appeared probable to the authors that, with a proper adjustment of the concentration, even the quantitative difference could be eliminated. They concluded that deep lung irritants preferentially attacked the respiratory bronchioles because of delayed clearance in that region.

In 1920, Underhill [31] exposed dogs to phosgene and noted the development of pulmonary edema which was maximal at 24-36 hours and resolved in animals surviving 10 days or more. He concluded that the minimum lethal concentration of phosgene for dogs was 310-350 mg/cu m (75-87 ppm). He found that dogs that survived for 3 days usually recovered. He also concluded that recovery from gassing increased the likelihood of death from regassing of dogs, which differs from the findings of Box and Cullumbine [40] in rats. Underhill explained that tolerance is demonstrable only with low concentrations; it does not decrease subsequent reactions to lethal concentrations. Winternitz et al [48] presented detailed information on the pathology found in these animals at autopsy.

Long and Hatch [29] reported that a reduction in the rate of respiratory uptake of carbon monoxide was an early and sensitive test of pulmonary impairment following exposure to pulmonary irritants. The test was developed using unanesthetized rats and phosgene as the test irritant. The animals were exposed to phosgene for 30 minutes at the following levels: 0.5-1, 1-2, 2-3, 3-4, and 4-5 ppm. The responses included a decrease in pulmonary uptake in CO which was progressive for 6-8 hours. followed by gradual recovery. They found that their test detected changes even at the lowest level of phosgene exposure (0.5-1 ppm) in the absence of microscopic changes at autopsy.

Boyd and Perry [30] exposed rabbits to phosgene for 30 minutes at a concentration of 270 mg/cu m (67 ppm). They reported a latent period of several hours following exposure. After the latent period, pulmonary edema developed.

Noweir et al [49] exposed rats to decomposition products of carbon tetrachloride at its TLV (10 ppm) and demonstrated [9] that up to 10 ppm of phosgene could be produced by thermal decomposition of this level of carbon tetrachloride. Thermal decomposition was achieved by passing a stream of carbon tetrachloride over a variety of hot surfaces including iron and glass, as well as open flames. Animals were exposed for 12 or 60 minutes to phosgene at concentrations of 10 or 2 ppm allowing an equal Ct of 120 ppm-minutes. Mixtures of decomposition products were tested as well. No marked potentiation of each irritant's effects upon the others was discovered. They found that chlorine, chlorine dioxide, and hydrogen chloride as well as phosgene contributed to respiratory damage.

Winternitz et al [50] studied the comparative pathology of acute phosgene poisoning. They reported that the pathologic findings of acute phosgene poisoning were similar in goats, dogs, monkeys, rabbits, guinea pigs, rats, and mice. These findings consisted primarily of pulmonary edema which increased in severity with the length of survival of the species. The most susceptible species, monkeys and guinea pigs, died prior to the development of pulmonary edema as severe as that seen in the dog or goat.

The basic mechanism of action by which phosgene produces lung damage has, as yet, not been established. The original supposition that liberated HCl was the toxic agent was never proved. A number of experiments carried out in World War II appear to have disproved the liberated HCl hypothesis and shown that phosgene affects tissues because the carbonyl group combines with free amines of cell enzymes or other critical substances. [51] A more recent theory is that of Ivanhoe and Meyers [52] who exposed rabbits to

phosgene at concentrations ranging from 50 ppm for 14 minutes to 200 ppm for 25 minutes. Their results showed a marked decrease in sympathetic nervous system activity in exposed animals. The authors concluded that phosgene toxicity was an example of acute pulmonary edema resulting from a hypoactive-sympathetic or neuroparalytic state in the host. This is corroborated, in part, by the work of Frosolono [53] who studied rat lungs by electron microscopy after exposures of 1,000 ppm-minutes to 4,320 ppm-minutes. The author noted interstitial edema as the common denominator of phosgene poisoning and felt that the autonomic nervous system might indeed play a significant role.

Cameron and Foss [54] exposed a group of animals to phosgene at an average concentration of 4.38 mg/cu m (1.1 ppm) for 5 hours/day for 5 days. The animal exposure group consisted of 20 mice, 10 rats, 10 rabbits, 2 cats, and 2 goats. After 24 hours, 50% of the mice were dead (10/20); after 48 hours, another 8 died, resulting in a casualty rate of 90% in 48 hours (18/20). All mice showed marked mottling of lungs with congestion, edema, and what was described as emphysema. Two rabbits died after 48 hours (2/20). On examination, one showed large areas of collapse in the lung with congestion and edema. The other rabbit showed some edema and congestion. The remaining animals survived and were killed at the end of the 5 days of exposure. Microscopic examination of the lungs of 37 of the animals showed that 22 (59%) had lung changes graded as severe, 15 (41%) had mild lung changes. Severe lesions were found in the cat, rabbits, guinea pigs, and mice. Goats and rats were much less affected. Edema was present in 35 of the 37 examined (95%), with severe edema in 12 animals, moderate edema in 13, and slight edema in 10. All species showed some degree of edema.

In a subsequent study, Cameron et al [55] exposed a group of animals to phosgene at an average concentration of 3.47 mg/cu m (0.86 ppm) for a single 5-hour exposure. The animal exposure group consisted of 20 mice, 10 rats, 10 guinea pigs, 10 rabbits, 2 cats, 2 monkeys, and 2 goats. On the morning following exposure, 10% of the rats (1/10) and 60% of the mice (12/20) were dead. There were no other casualties, although one cat and one monkey were very ill with considerable labored breathing. All survivors of the experiment were killed on the morning following exposure. All animals were then autopsied and one lung from each animal was fixed in formalin for sectioning. Upon examination, 54 out of 56 animals (96.4%) showed microscopic evidence of pulmonary involvement which was severe in 29 animals (39%), mild in 17 (31%), and slight in 16 (30%). The most frequent lung change noted was edema.

In another study, Cameron et al [56] reported the results of exposing a variety of animals to phosgene at an average concentration of 0.9 mg/cu m (0.2 ppm) for 5 hours daily for 5 consecutive days. The experimental group consisted of 20 mice, 10 rats, 10 guinea pigs, 10 rabbits, 2 cats, and 2 goats. No deaths occurred during the exposures. Except for some labored breathing noted in the cats and in one goat, the other animals showed little evidence of distress. At autopsy, pulmonary lesions were seen in 67% of the animals. In the opinion of the investigators, the great majority of such lesions were slight and of little significance. Discounting the more susceptible animals (guinea pigs) and correcting for the normal incidence of disease in laboratory animals, the authors estimated that probably between 5 and 10% of the animals showed moderately severe lesions. Pulmonary edema was noted in 41% of the animals but was

considered to be slight in most cases. In 6 animals (1 rabbit, 1 mouse, 1 rat, and 3 guinea pigs), it was extensive. Acute bronchitis was noted in 22% of the animals and bronchial regeneration in 20%. Their results are shown in Table III-3.

In their summary statement, the authors [56] advanced the opinion that there is little doubt that repeated exposure at low concentrations (0.9 mg/cu m) induces damage to the lungs but that such damage was rarely severe. Seemingly in contradiction with this, they also stated that, at this concentration, some fairly severe changes are found in the lungs of experimental animals.

TABLE III-3

SEVERITY OF LUNG LESIONS AFTER EXPOSURE TO 0.2 PPM PHOSGENE, 5 HOURS DAILY FOR 5 CONSECUTIVE DAYS

	Goats	Cats	Rabbits	G. pigs	Rats	Mice	Total	%
Total Number of Animals	2	2	10	10	10	20	54	
Severe lesions	0	0	0	1	1	0	2	4
Mild lesions	0	0	1	3	1	1	6	11
Very slight lesions	0	1	5	6	3	13	28	52
No lesions	2	1	4	0	5	6	18	33
Incidence of pulmonary edema	0	1	5(1)	7(3)	2(1)	7(1)	22	41
Incidence of severe bronchitis	0	1	5	5	1	0	12	22
Incidence of bronchial regeneration	0	0	4	5	1	1	11	20
Incidence of broncho-pneumonia	0	0	0	1	1	0	2	4

Figures in parentheses under pulmonary edema indicate number of animals showing fairly severe edema.

From reference 56

Correlation of Exposure and Effects

Phosgene is known historically as a respiratory poison used to disable large masses of soldiers. It is no longer used as a military weapon but has become an important industrial chemical. The focus of impairment to the health of those who are exposed to high concentrations of the gas has therefore shifted from the military to industry. Epidemiologic studies [38, AF Myers, written communication, November 1974] have shown no ill effects definitely attributable to phosgene in workers exposed to phosgene at an average of 0.125 ppm or less for considerable periods. However, the investigations of Levina and Kurando [38] did not mention studying the possibility of pulmonary disease, and the Myers communication indicated that, most of the time, levels were actually much lower than 0.125 ppm.

Animal studies, for the most part, have attempted to duplicate the war gas or accidental overexposure situation where there is exposure to phosgene at high concentrations for relatively short periods. These studies, summarized in Table III-4, [29,30,31,39,40,41,42,45,46,48] have shown a fairly similar picture, ie, animals dying immediately show severe pulmonary epithelial and capillary destruction, and animals surviving show variable amounts of bronchiolitis, pneumonitis, bronchial plugging, atelectasis, pulmonary consolidation, pneumonia, and emphysema.

The one animal study [56] devoted to long-term, repeated exposure to phosgene at low concentrations produced pulmonary edema in 41% of the animals. After correcting for the normal incidence of disease in laboratory animals, 5-10% of the animals had moderately severe lesions.

Human exposures to phosgene reported in the literature (summarized in Table III-5) consist of many instances of acute overexposure. The work of Galdston et al [28,35] is the best substantiated in the American literature and gives details of pulmonary function studies in 11 workers with single acute or repeated exposures to phosgene at unknown concentrations. Unfortunately, other pulmonary irritants were sometimes involved.

Galdston et al [35] gave evidence that repeated exposures to phosgene can result in residual pulmonary problems. This paper reports on 5 workers who were studied in detail, 2 of whom had abnormal chest films. It is difficult to extrapolate the results found in these workers to what might be expected in the general population of workers exposed to phosgene over long periods of time.

Levina et al [36] found no pulmonary abnormalities in Soviet workers exposed to 1-2 mg/cu m of phosgene for over a 6-month period. Other inhalants apparently caused hematologic abnormalities. Unfortunately, no mention is made of any pulmonary function studies done on these workers.

In summary, there are no truly pertinent data in the scientific literature concerned with long-term effects on humans exposed to phosgene at low concentrations. Animal data show a 5-10% incidence of severe pulmonary problems in animals exposed at 0.2 ppm. Despite Cherkes' [26] statement that the dog is the animal most resembling man in terms of susceptibility to phosgene, neither Cherkes nor any other investigator has offered any concrete data to support this contention. In fact, Winternitz et al [50] concluded that, based upon pathologic findings, phosgene lung changes were basically similar in all the species studied. Referring to investigations of others, Cucinell [57] has stated that, at least in terms

of lethality, man is about as susceptible as the mouse. But he also pointed out that data with which to correlate the toxicity of phosgene in man to that in laboratory animals at low concentrations do not exist.

Carcinogenicity, Mutagenicity, and Teratogenicity

Data on other possible effects of toxic chemicals, such as carcinogenicity, mutagenicity, or teratogenicity have not been reported for phosgene, and there is no analogy on which to postulate such effects on long-term, low-level exposure. However, with the likely ability of phosgene at high concentrations to cause extensive damage to lung tissue, it is conceivable that among survivors of such exposures occasional neoplasia might occur as the consequence of regeneration of damaged tissue.

TABLE III-4

PHOSGENE INHALATION EXPOSURES AND EFFECTS--ANIMALS

Authors	Exposure Variables	Exposure Time	Effects
Frosolono [53]	Rats 1,000-4,320 ppm-min		Interstitial edema
Ivanhoe & Meyers [52]	Rabbits, 50-200 ppm	14-25 min	Decrease in sympathetic tone
Underhill [31] as reported by Winternitz et al [48]	Dogs, 44-120 ppm	30 min	Pulmonary edema, pneumonia, emphysema, death
Box & Cullumbine [40]	Rats 20 ppm	10 min	*
	Rats 55-100 ppm	"	Reduction in death rate from 74% to 33% by previous challenge
Durlacher & Bunting [46]	Dogs, 72 ppm	30 min	Pulmonary consolidation, death
Boyd & Perry [30]	Rabbits, 67 ppm	30 min	Pulmonary edema
Clay & Rossing [39]	Dogs 24-40 ppm	30 min 1 or 2 exposures at rate of 1-3/week	Acute bronchiolitis
	"	30 min 4-10 exposures at rate of 1-3/week	Chronic bronchiolitis
	"	30 min 30-40 exposures at rate of 1-3/week	Emphysema
Cordier & Cordier [44]	Cats and guinea pigs 2.5-6.25 ppm	10 min/day x 2-41 days	Pulmonary edema, bronchitis, bronchopneumonia, death

* Animals were gassed to determine effect of pregassing upon a later challenge

TABLE III-4 (CONTINUED)

PHOSGENE INHALATION EXPOSURES AND EFFECTS--ANIMALS

Authors	Exposure Variables	Exposure Time	Effects
Long & Hatch [29]	Rats, 0.5-5 ppm	30 min	Decreased pulmonary CO uptake
Rinehart & Hatch [41]	Rats, 0.5-4 ppm	5-480 min	Increased resistance to breathing decrease in diffusion capacity
Gross et al [42]	Rats, "	"	Chronic pneumonitis
Cameron & Foss [54]	Variety of animals, 1.1 ppm	5 hours/day x 5 days	Pulmonary edema, death
Cameron et al [55]	Variety of animals, 0.9 ppm	5 hours/day x 1 day	Pulmonary edema, death
Cameron et al [56]	Variety of animals 0.2 ppm	5 hours/day x 5 days	Pulmonary edema
Koontz [45]	Dogs, unknown	Unknown	Bronchial plugging and atelectasis

TABLE III-5

PHOSGENE INHALATION EXPOSURES AND EFFECTS--HUMANS

Authors	Exposure Variables	Exposure Time	Effects
Theiss & Goldmann [19]	a) Unknown	Brief	Pulmonary edema
	b) 1 mole of phosgene**	"	"
	c) Unknown	30 min	Pulmonary edema, death
Gerritsen & Buschmann [6]	a) " **	Indefinite	"
	b) " **	3 hours	Pulmonary edema
Spolyar et al [5]	Unknown (15 ppm)*, **	<3 1/2 hours	Pulmonary edema, death
Glass et al [21]	Unknown **	4 1/2 hours	Acute bronchitis
Everett & Overholt [23]	"	Brief	Pulmonary edema
Delepine [25]	a) Unknown	"	Bronchial irritation, death
	b) "	"	"
English [8]	Unknown **	8 hours	Bronchiolitis, reactivation of a duodenal ulcer
Seidelin [7]	Unknown **	Brief	Pulmonary edema
Stavrakis [27]	a) Unknown	"	"
	b) "	"	Pulmonary edema, death
Steel [34]	a) Unknown	"	Acute bronchitis and delirium
	b) "	"	Pulmonary edema and delirium
Derrick & Johnson [22]	Unknown **	"	Pulmonary edema, death

* Re-created exposure simulating accident

** Simultaneous exposure to chlorinated hydrocarbons

IV. ENVIRONMENTAL DATA

Environmental Concentrations and Engineering Controls

Reports of workroom air levels of phosgene in industrial processes utilizing phosgene as a starting material have not been published in the United States. In contrast, the formation of phosgene by decomposition of chlorinated hydrocarbons and the resulting occupational exposures have been extensively studied. In evaluating ventilation plans for the control of phosgene in a plant conducting "phosgenation," the Bureau of Engineering Safety, Department of Labor and Industry, State of New Jersey, [24] reported briefly on workroom air concentrations of phosgene. Air sampling in the breathing zones of chemical operators during routine phosgenations showed concentrations of phosgene "well within the TLV of 0.1 ppm" at the time of this investigation. Details of the sampling and analysis procedures were not given, nor were individual sampling results reported. Although management had filed plans for canopy hoods over the reactors, the ventilation actually consisted of flexible ducts, 4 inches in diameter, to each kettle. Engineering controls did not conform to the applicable MCA Chemical Safety Data Sheet. [1] Over a 2-year period prior to this investigation, at least 6 employees had been acutely exposed to unknown concentrations of phosgene. [24] Four of these were not able to return to work the day after exposure. One death had occurred and was attributed to phosgene inhalation. No details were reported. After the fatality, the Bureau of Engineering Safety ordered all phosgene operations stopped because of deficient controls. Subsequently, management of the plant discontinued the use of phosgene.

In 1962, Filatova et al [58] investigated health hazards occurring in the manufacture of diisocyanates utilizing phosgene as one of the raw materials. The authors commented favorably on the isolation of hazardous sections of the plant as a method of control. Local exhaust ventilation was utilized extensively. Ninety-one workroom air samples were taken and analyzed for phosgene by undescribed methods. Units of analytical results were not stated but are assumed to be mg/liter. Results showed phosgene concentrations ranging from a reported 0 to 0.013 mg/liter (0-3.25 ppm), with the most frequently observed concentrations ranging from a reported 0 to 0.0004 mg/liter. Of the 91 samples, 31 were negative for phosgene. However, an unspecified number exceeded the Maximum Permissible Concentration (MPC) of 0.5 mg/cu m (0.125 ppm) during such operations as cleaning vats or distillation apparatus. Recommendations for control included mechanization of manual procedures, provision of independent ventilation systems for each floor, limiting the number of hoods for each system to 10-12, and providing an intake velocity of at least 1-1.5 meters/second (197-296 feet/minute) for hoods with movable sash and louvers.

In 1966, Filatova [59] reported industrial hygiene evaluations of phosgene in the production of isocyanates. A continuous-flow method was used, involving the use of remote control equipment and the mechanization of unspecified operations, and so-called hermetization which was assumed to mean enclosure or sealing of equipment, particularly that operating under pressure. Under these conditions, 91% of air samples were below maximum permissible concentrations for phosgene. Filatova did not further report the results of air sampling.

Also in 1966, Levina et al [36] reported on industrial hygiene problems and worker health in monuron production. Monuron was defined as 3-(p-chlorophenyl)-1,1-dimethylurea and was used as an agricultural pesticide. Monuron was produced by the interaction of parachlorophenylisocyanate and dimethylamine. Synthesis of the isocyanate involved the use of phosgene. The authors criticized the plant layout because units using materials of high toxicity (eg, phosgene) were located throughout the general work area rather than being isolated. Local exhaust ventilation placed at sources of contaminant escape, in addition to general ventilation, provided some control. Makeup air inlets were considered by the authors [36] to be wrongly placed. Workroom air samples analyzed for phosgene (503 samples) showed concentrations commonly ranging from 1.0 to 2.0 mg/cu m (0.25-0.5 ppm). The maximum permissible concentration in Russia at that time was 0.5 mg/cu m (0.125 ppm). Most phosgene was released during disassembly and repair of pumps, which often became clogged. This operation, as well as repair of other phosgene equipment, the transmission of phosgene under pressure, and the taking of samples for quality control were carried out by workers wearing gas masks. Released phosgene was said to be neutralized with ammonia. Control recommendations included replacement of equipment with improved hermetically sealed pumps and revamping of the makeup air system to provide uniform air supply at low velocities. Mechanization of systems, relocation in areas removed from the general workrooms, and use of remote controls were also recommended.

In 1967, Levina and Kurando [38] described exposures to phosgene and other chemicals in the manufacture of isopropyl phenylcarbamate, used as a herbicide. Totally enclosed equipment kept under reduced pressure

prevented the escape of toxic chemicals to a large degree. Equipment and connections were made of corrosion-resistant materials, with special attention to flanges and valves. Atmospheric pollution was prevented by trapping gases and returning them to process. However, where manual labor was substituted for mechanization or total enclosure, some gas escape was possible. Hazardous processes were separated, and ventilation was reported to be correctly designed and located. Makeup air was supplied. Phosgene was detected in 30% of all air samples taken; of the samples where phosgene was detected, the usual concentration was 0.5 mg/cu m (0.125 ppm), the maximum permissible concentration. Methods of sampling and analysis were not described. During operations such as quality control sampling, pressure transfer of phosgene, and pump repair, the concentration of phosgene in the workroom air was 1.0 mg/cu m (0.25 ppm). However, these concentrations were described as transient, because the premises were cleared by release of ammonia whenever the odor of phosgene became discernible. Gas masks were worn by personnel engaged in these tasks. Recommendations for further control included improved sealing of equipment, elimination of manual operations by maximum automation, use of protective clothing, and good personal hygiene.

Rispoli [60] reported 1 and 5 ppm phosgene in a plastics-manufacturing plant. No details of sampling or analysis were given, but ventilation was described as insufficient.

Reporting on 109 cases of phosgene inhalation over a 12-year period, Thiess and Goldmann [19] selected the 3 most severe cases for more detailed discussion. None of the exposures reported were long-term inhalation of low concentrations but were acute accidental exposures at relatively high

concentrations. No determinations of workroom air concentrations were reported, but in 2 of the severe cases some assumptions and calculations were made which might have defined the exposure level involved.

One case described by Thiess and Goldmann [19] involved a worker removing brick lining, presumably saturated with phosgene, from a reaction vessel. The chemical process for which the reactor was constructed was the chlorination of aluminum oxide in the presence of carbon monoxide for the production of aluminum chloride. Phosgene was a byproduct of this reaction. Contrary to instructions, the brick lining was removed with a jackhammer from the inside of the furnace and without respiratory protection. Instructions had been given to remove the brick from the outside of the vessel and to wear respiratory protection. The exposure lasted about 30 minutes, during which the worker was presumed to have inhaled dust particles with adsorbed phosgene. Again, assumptions and calculations based on porosity measurements of the brick indicated that a total volume of 2.5 liters was available for absorption of gas by the lining material. This exposure terminated fatally. There is at least a suggestion that the effect of phosgene was enhanced because of its association with particulate matter, but this was not proved.

Unpublished data show that air concentrations of phosgene were generally low in a chemical manufacturing unit where phosgene was used as a raw material. (AF Myers, written communication, November 1974) Air samplers placed on operators showed concentrations on analysis ranging from nondetected to 0.08 mg/cu m. Samples taken in fixed locations ranged from nondetected to 0.52 mg/cu m. During the 2-month period of fixed location sampling, there were 5 instances of off-scale measurements (greater than

0.55 mg/cu m) reportedly due to leaks. In summary, the workroom air measurements of phosgene in a manufacturing unit showed that, about 25% of the time, operators were exposed to very low but measurable concentrations of phosgene. Sampling procedures used did not determine individual peaks. Details of controls or of fixed sampling locations were not given. The plant surveyed was reportedly old and built with out-of-date technology.

The decomposition of chlorinated hydrocarbons by means of heat or ultraviolet radiation with the formation of phosgene has been blamed for a number of incidents involving respiratory tract damage in humans. [5,6,7,21,61,62,63,64, written communication from DR Parker, Oregon State Board of Health, November 1964] This has stimulated a great deal of investigation of the decomposition products formed when chlorinated hydrocarbons are exposed to ultraviolet radiation and heat, such as open flames and burning cigarettes. The following exemplify these investigations.

In 1933, the Underwriters Laboratories [65] reported the results of decomposition of a number of chlorinated hydrocarbons (ie, chloroform, carbon tetrachloride, a number of halocarbon compounds, dichloroethylene, methylene chloride, and others) exposed to gas flames, hot electric range units, oil fires, wood fires, and hot metal surfaces. The temperatures of these heat sources were not specified. Decomposition products were analyzed for hydrogen chloride, phosgene, chlorine, and others. The analytical method for phosgene involved the reaction of phosgene with aniline to form diphenylurea. The influence of relative humidity and initial concentration of chlorinated hydrocarbon was studied. The major decomposition product found was hydrogen chloride measured as total acids,

which ranged from nondetected to 8.86 volume percent, followed by phosgene with concentrations ranging from a reported zero to 0.143 volume percent. Chlorine was found in the concentration range of a reported zero to 0.164 volume percent.

In 1936, Yant et al [66] found significant quantities of phosgene in the decomposition products of carbon tetrachloride exposed to excelsior fires and heated steel surfaces. The analytical method used was based on the formation of diphenylurea which was found by Yant et al [66] and the Underwriters Laboratories [65] to be essentially free from interference by hydrogen chloride and chlorine. More recent research has shown that hydrogen chloride and chlorine do significantly interfere with the diphenylurea analytical method. [67]

Elkins and Levine [68] investigated the decomposition of halogenated hydrocarbons when passed through burning cigarettes and cigars. Analytical methods used were for total chloride and therefore not specific for phosgene. However, based on the small amounts of total chloride found in the samples, the authors [68] concluded that phosgene was not a hazard when cigars or cigarettes were smoked in the presence of chlorinated hydrocarbon vapor.

In 1955, Little, [20] using the p-dimethylaminobenzaldehyde and diphenylamine method, confirmed the conclusions of Elkins and Levine [68] that phosgene was not formed in sufficient quantities by smoking to present a hazard. He also found that various chlorinated hydrocarbons in contact with a heated silica tube did not produce significant quantities of phosgene below 400 C. Even in atmospheres containing small amounts of phosgene, finding no detectable phosgene in effluent gas from cigarettes

led to the conclusion that, if any phosgene were formed, it was subsequently decomposed when passed through the combustion zone of the cigarette. It was also hypothesized that the phosgene was absorbed by the tar fraction of the tobacco combustion product.

Crummett and Stenger [69] found that, in contact with heated metals, methyl chloroform (1,1,1-trichloroethane) produced relatively small amounts of phosgene, but much more hydrogen chloride than did carbon tetrachloride.

Dahlberg, [70,71] Dahlberg et al, [72] and Dahlberg and Myrin [73] extensively investigated the interaction of ultraviolet radiation with chlorinated hydrocarbons. These studies indicated that relatively lower amounts of phosgene were formed compared to the quantities of dichloroacetyl chloride formed simultaneously. Although there have been no environmental limits recommended for dichloroacetyl chloride, it seems likely that it would be a strong irritant to skin and eyes. Actual measurements of decomposition products of trichloroethylene in welding shops confirmed their experimentally derived conclusions. In 10 welding shops under varying conditions of ventilation and trichloroethylene concentration, phosgene was found in concentrations ranging from 0.003 to 3.0 ppm, while dichloroacetyl chloride concentrations ranged from less than 0.01 to 13.0 ppm.

In a more recent investigation, Andersson et al [74] experimentally studied phosgene formation from perchloroethylene (PCE) during welding. Welding was carried out in a closed chamber, into which varying concentrations of PCE had been introduced by evaporation of the solvent in front of a fan. Analysis for both perchloroethylene and phosgene were done by gas chromatograph with an electron-capture detector. The authors

concluded that welding in air containing PCE below its threshold limit value of 100 ppm was more hazardous than welding in air containing trichloroethylene or methyl-chloroform because of the much faster formation of phosgene from PCE. Levels of perchloroethylene ranged from 2.1 to 30 ppm, while phosgene formed after 5 or 10 seconds ranged from 0.2 to 1.7 ppm.

Noweir et al [75] found that, at very high temperatures, phosgene itself decomposes, perhaps accounting for concentrations lower than expected. However, phosgene decomposition was greatly reduced in the presence of carbon tetrachloride, indicating that, at least for this chlorinated hydrocarbon, phosgene formed by its decomposition should be relatively stable as long as some carbon tetrachloride remained.

A number of occupational exposures to decomposition products of chlorinated hydrocarbons have been reported. In 1947, Hill [64] briefly reported an investigation of complaints of workers exposed to fumes arising from a carbon arc-welding operation. A presumably adequate exhaust ventilation system existed, but the fumes causing the complaints were sometimes strong enough to be detected at some distance from the arc. The workers had not complained prior to the relocation of the operation to a room near a trichloroethylene degreaser. Qualitative tests indicated the presence of phosgene presumably caused by breakdown of trichloroethylene vapor by the ultraviolet radiation of the arc. It was recommended that the degreaser be replaced with alkaline wash equipment and that the use of chlorinated hydrocarbons in the welding room be prohibited.

Spolyar et al [5] investigated the circumstances causing the death of an employee who had installed a trichloroethylene degreaser and had

operated it without the condensing coils functioning. A strong odor of solvent was present in the room when the employee's body was discovered. An investigation by the authors [5] took place 3 weeks later, disclosing the following:

(1) Trichloroethylene had been used in a degreaser designed for higher-boiling perchloroethylene, causing excessive loss of solvent.

(2) A fuel-oil-burning space heater was located in the degreaser room, and all windows and doors had been closed on the day of the accident.

(3) Under test conditions, 225-450 ppm trichloroethylene were found near the degreasing tank.

The trichloroethylene vapor passing through the firebox of the space heater could be decomposed with the possible formation of phosgene. A re-creation of the situation at the time of the accident, described in Effects on Humans, was undertaken, during which it was noted that one of the solvent heaters in the degreaser became red-hot. Samples taken at that time in the approximate location of an operator's breathing zone showed much higher levels of trichloroethylene and 15 ppm phosgene. The authors [5] suggested that the decomposition of the trichloroethylene was caused by the degreaser heating element rather than by the space heater, although a back pressure in the smoke pipe of the latter could occur under adverse weather conditions because of the configuration of the stack.

Although some effects due to trichloroethylene were apparent, the authors concluded that the cause of death was consistent with phosgene exposure.

In 1951, Wulfert [62] reported the results of an investigation of the manufacturing of hard metal drills (presumably with tungsten carbide drill

inserts). Drills were washed in trichloroethylene after which the solvent was blown off with compressed air into the shop atmosphere prior to brazing. Brazing was completed with an induction coil or gas brazing, depending on the size of the drill. Results of workroom air sampling for phosgene were reported only as exceeding 1 ppm. The author [62] concluded that local exhaust ventilation was required.

An investigation of worker complaints was reported by Challen et al [76] in 1958. The workers involved were employed in a welding shop fabricating aluminum milk churns. Upper respiratory symptoms were thought to have been caused by phosgene from decomposition of trichloroethylene originating from a tank of the solvent located in an adjacent bay. Air samples were taken and analyzed for trichloroethylene, ozone, and phosgene. All phosgene samples contained less than 0.1 ppm, although the trichloroethylene concentrations ranged from a reported 0.0 to 238 ppm and the ozone concentrations ranged from 0.9 to 1.7 ppm. The authors [76] concluded that ozone rather than phosgene was the source of the complaints. Exhaust ventilation was installed in the degreasing shop and at the welding positions and operating procedures to prevent solvent drag-out were implemented, resulting in improved working conditions.

In a November 1964 letter from DR Parker to J Boyer, the Oregon State Board of Health reported finding a phosgene concentration in air of 4.86 mg/cu m (1.2 ppm) in the vicinity of welders who had complained of noxious odors. Two other samples indicated no phosgene. Decomposition of perchloroethylene vapor from a recently installed degreaser was considered to be the source of the phosgene. The perchloroethylene concentration on the day of the high phosgene sample was as much as 37 ppm. Remedial

recommendations included relocation and isolation of the degreaser from the welding area. A ventilated booth was suggested for this purpose.

In summary, the earlier experimental studies [65,66] clearly indicate phosgene produced by decomposition of chlorinated hydrocarbons could present a hazard; there are cases that clearly show phosgene present in sufficient quantity to be a health hazard. [5,65,66] It should be noted that the various methods of heating, different temperatures used when producing the chlorinated hydrocarbon decomposition products, and the different analytical methods probably accounted for much of the variation from study to study.

Sampling and Analytical Methods

Detection methods for phosgene prior to the mid-1950's met minimal demands for sensitivity and detection limits. Soon after this, however, large-scale industrial use of the gas in polymer manufacturing processes prompted increased emphasis on potential industrial hygiene problems associated with its use. Linch et al [77] reviewed most of the analytical methods for phosgene used prior to 1965 and classified them into (1) physical measurement, (2) determination of chloride ion after hydrolysis, (3) iodometric procedures, (4) gravimetric procedures, and (5) colorimetric procedures.

Physical measurements were based on the olfactory or taste response imparted to tobacco smoke immediately after phosgene inhalation and was considered to be a very sensitive indicator [77] of the gas, although it suffered from the obvious disadvantage of depending on the exposed worker to determine his exposure.

A method of analysis based upon chloride present after hydrolysis was reported [77,78] to depend on aqueous decomposition of phosgene in dilute alkali. Residual base was titrated or the chloride present was determined by the Volhard or the bichromate method; the hydrogen chloride gas produced upon hydrolysis could also be absorbed in ammoniacal silver nitrate and the resultant silver chloride determined gravimetrically. [79,80] However, these procedures were susceptible to physiologically inert, acid-reacting, or chloride-producing components occurring simultaneously with the phosgene. [77]

Iodometric procedures in anhydrous acetone with thiosulfate titration suffered from inherent field limitations due to the anhydrous conditions necessary and evaporation of the volatile solvent. [77]

Gravimetric procedures included (1) a method based on insoluble diphenylurea derived from phosgene and aniline, but which was sensitive to halogen interferences [80] and limited to a lower detection threshold of about 10 ppm [77]; and (2) a modified Kjeldahl nitrogen analysis of the diphenylurea which offered little improvement.[77]

Review of colorimetric analytical methods [81,82,83,84] that were developed indicated that the most sensitive and specific detector was 4-(4'-nitrobenzyl)pyridine (NBP) plus N-benzylaniline although it did not readily lend itself to a liquid reagent system. Later investigators [85] developed a method using diethylphthalate as an absorber with NBP and N-benzylaniline and easily determined phosgene at concentrations less than 0.1 ppm in air. Spot tests have been described [77,79,81] for phosgene which generally rely on colored complex formation between heavy metals and diphenylcarbazones.

Various detector papers and treated crayons have been described for determination of phosgene [86,87,88,89] with the most sensitive prepared from a benzene solution of 2% 4-(4'-nitrobenzyl)pyridine, 5% N-phenylbenzylamine, and a chalk matrix which was then dried and pressed into crayons and was capable of detecting 8 ppb phosgene in air after a 1-minute exposure. All of the crayon and detector paper methods suffer sensitivity loss from storage; color change sensitivity to oxygen, chlorine, hydrogen chloride, and daylight; and the inherent disadvantage of reliance on visual detection of small color differences. [88, 89]

Direct reading, colorimetric detector tubes are available for the measurement of phosgene. [1,2,90,91,92] They offer a quick and easy method of determining approximate quantities in the reported range of 0-50 ppm and of indicating the need for initiation of emergency procedures or for further, more accurate evaluation. Today, most detector tubes are of the "length of stain" type. A fixed volume of air is drawn through a glass tube containing a solid sorbent which reacts with phosgene. A color stain is produced which varies in length with the concentration measured. The length of stain is compared with a calibration chart provided by the manufacturer. Accuracy expected is only $\pm$ 30%.

An ultraviolet technique was developed [93] which uses the absorption at 254.5 nm of 1,3-diphenylurea formed when phosgene reacts with aniline-saturated water. A later refinement of the technique [67] was made using an extraction step with a 1:1 solution of n-hexane and 1-pentanol. The latter method allowed detection of 0.01 ppm phosgene in air with a 30-liter air sample. Collection efficiency was only about 90%.

A micromanometric method was described [94] which utilized measurement of gaseous reaction products resulting from hydrolysis of phosgene, but details as to sensitivity and specificity were lacking.

Gas chromatographic techniques provide the most specific and sensitive detection methods for phosgene. A silica gel column at 56.5 C with a hot wire detector was first used to detect phosgene in pyrolysis products on a semiquantitative basis. [95] The development of the electron capture detector for the gas chromatograph provided a high degree of specificity for phosgene. This detector was first tried [96] in conjunction with a dodecylphthalate column at 50 C with resultant 2% standard deviation and detection down to 1 ppb. The authors suggested that this method can possibly be used as the basis for an automatic, continuous monitoring system. A later refinement [97] used a column of "20% silicon oil DC 200" (probably silicone DC 200) on Chromosorb W at 25 C for detection from below 1 ppb to 0.1 ppm phosgene. Jeltes et al [98] confirmed detection below 0.005 ppm but pointed out inherent difficulties due to hydrolysis by trace amounts of moisture, leakage of transfer syringes, and problems in transporation and storage of the air samples. Basu et al [99] presented operational parameters, retention times, and response factors for gas mixtures including phosgene using a 3-column, 3-detector (thermal conductivity) gas chromatographic system with a resultant reproducibility represented by a ±1% standard deviation.

Although the gas chromatographic methods display excellent analytical capabilities, field use is hampered by the lack of an adequate personal sampling method. Recent investigations by Barrett et al [100] of a number of solid sorbents indicated that phosgene can be efficiently collected with

activated carbon, alumina, and porous glass, but no satisfactory desorption technique was found.

Methods have been reported for automatic or continuous monitoring of phosgene in air. [77,90,96,101,102,103, RW Miller, written communication, May 1975] The instrumentation requirements make the methods impractical for field use. In-plant applications may be possible but the lower limit of detection may be questionable.

One continuous-sensing instrument which has been found to be generally reliable below 0.1 ppm of phosgene is the Army's M8 portable alarm device for toxic chemical agents. It uses an electrochemical cell as the sensing unit and is designed to give a warning signal when the concentration of phosgene exceeds a certain value. The instrument is therefore not ideally suited for measurement of varying concentrations, but may well have some application as a monitoring or safety device. The device has been tested at concentrations of 0.074 ppm, 0.185 ppm, 0.443 ppm, and 0.625 ppm. The response was found to be linear over this range. [100]

The method of choice for detection of phosgene in the workroom air should provide for a minimum limit of detection of less than 0.05 ppm, reasonable freedom from interference from commonly expected chemicals (see Appendix II) and should permit relatively easy sample collection. The method which best meets these requirements is described in an Analytical Guide by the American Industrial Hygiene Association [104] and is an improved nitrobenzylpyridine colorimetric method. It involves drawing air through a midget impinger containing 10 ml of a solution of 0.25% 4-(4'-nitrobenzyl)pyridine and 0.5% N-phenylbenzylamine in diethylphthalate and

measurement of the subsequent color development at 475 nm. Sampling efficiency is at least 99%, [85,104] and the range 0.05-1.0 ppm can be measured if a 25-minute sample at 1 liter/min is collected in 10 ml of reagent. No statistically significant interferences were observed [85,104] from carbon tetrachloride, chloroform, perchloroethylene, trichloroethylene, dichlorodifluoromethane, chlorine, hydrogen chloride, or chlorine dioxide. Color stability is good (only 10-15% loss in color density after 8 hours), sampling and analysis equipment needed is simple and readily available, and operator expertise required is minimal.

V. DEVELOPMENT OF STANDARD

Basis for Previous Standards

In 1940, Bowditch et al [105] listed a maximum concentration of 1 ppm (4 mg/cu m) phosgene which was in effect in Massachusetts as a guide to manufacturers interested in maintaining satisfactory working conditions. In 1945, Cook [106] compiled a list of maximum allowable concentrations (MAC) of industrial atmospheric contaminants. Cook noted that 1 ppm (4 mg/cu m) was the MAC value for exposure to phosgene in the workroom air adopted by California, Connecticut, New York, Oregon, Utah, and the US Public Health Service. Cook referred to Fieldner et al [107] and Flury and Zernik [108] as a basis for the accepted value of 1 ppm (4 mg/cu m). Fieldner et al [107] indicated that 3 ppm of phosgene in air was irritating to the throat. Flury and Zernik [108] indicated that 1 ppm was the highest tolerable amount in man, with 1.25-2.5 ppm termed as dangerous to life if the exposure was prolonged.

In 1947, Bloomfield [109] reviewed the efforts of a committee within the American Conference of Governmental Industrial Hygienists to develop an MAC which could be adopted by all the states. Bloomfield [109] cited unanimous agreement on a phosgene MAC value of 1 ppm (4 mg/cu m) among 23 respondents.

In 1947, the American Conference of Governmental Industrial Hygienists [110] adopted an MAC value for phosgene of 1 ppm (4 mg/cu m). It was not stated if this MAC was intended as a ceiling value or as a TWA concentration. The April 1948 meeting of this same organization [111] adopted 1 ppm as a recommended limit and changed the name to Threshold Limit Values (TLV's).

The TLV of 1 ppm was considered "sufficiently low to cause no more than minimal effects" by the ACGIH according to the 1962 Documentation of the Threshold Limit Values. [112] The Documentation [112] cited Fieldner et al [107] and Henderson and Haggard [113] to explain its selection of a TLV of 1 ppm (4 mg/cu m) for phosgene. Fieldner et al [107] reported that the Chemical Warfare Service at the American University Experiment Station, Washington, DC, considered 1 ppm of phosgene (4 mg/cu m) the maximal concentration safe for prolonged exposure. Henderson and Haggard, [113] in their review on phosgene, referred to Fieldner et al [107] and listed 1 ppm (4 mg/cu m) or less as a maximum concentration allowable for prolonged exposure. Both sources listed 3 ppm as the concentration at which throat irritation first occurs. The Documentation did not quote any other experimental or occupational data to support its recommended TLV.

A change to 0.1 ppm (0.4 mg/cu m) was proposed by the ACGIH in 1964 [114] and 1965 [115] and adopted in 1966. [116] A TLV of 0.1 ppm (0.4 mg/cu m) was supported in the 1966 Documentation [117] because of the "seriousness of the response at the experienced levels of phosgene and by analogy with edemagenic agents of similar activity." The Documentation cited Gross et al [42] who wrote that phosgene, which could produce fatal pulmonary edema or acute chemical pneumonia at a high concentration, could also produce chronic pneumonitis at a low concentration. Gross et al suggested that the chronic pneumonitis is reversible even though residual pneumonitis could be found three months later. [42] The alveolar epithelium in rats can become irritated after a single 120-minute phosgene exposure at 0.5 ppm. [42] Studies by Box and Cullumbine [40] indicated that preliminary nonlethal doses of phosgene produced a transitory effect of

increasing tolerance to the gas. It was necessary to damage the lungs of rats and mice to produce this effect. Stokinger et al [118] reported chronic lung injury of small animals after repeated exposure to ozone. They stated that this is a separate process from the development of tolerance to subsequent acute exposure. In reviewing Stokinger's article, [118] the Documentation [117] stated that "the development of tolerance, however, is believed to be the triggering mechanism of chronic, irreversible pulmonary changes of emphysema and fibrosis from prolonged daily exposure at concentrations that produce no ostensible acute response." The Documentation [117] assumed that both ozone and phosgene caused pulmonary edema.

The 1971 Documentation [119] was the same as the 1966 Documentation [117] with a few minor style changes and one additional sentence listing a recommendation for the USSR (1959) of 0.1 ppm and mentioning Elkins, [120] who recommended 0.5 ppm. There was no discussion of the additional recommendations nor were any new references cited in the bibliography. In 1974, it was recommended [121] that the TLV of 0.1 ppm be changed to a ceiling concentration of 0.05 ppm (0.2 mg/cu m). Documentation for this change was published in the 1974 ACGIH Transactions [122] and refers to Stokinger's article on ozone toxicity studies [118] which indicated that there were chronic, irreversible pulmonary changes from prolonged daily exposure even though there was no acute response. None of the references cited specifically supported the ceiling limit of 0.05 ppm (0.2 mg/cu m) which was recommended because of the irritating effect of phosgene on the respiratory tract.

In 1971, Pennsylvania [123] adopted an environmental limit of 0.1 ppm (0.4 mg/cu m) for phosgene. It was a maximum average atmospheric concentration for an 8-hour day. A short-term maximum average exposure limit of 1 ppm (4 mg/cu m) for 5 minutes was established. Henderson and Haggard, [124] Imperial Chemical Industry, and Patty [125] were cited as the basis for the short-term limit in the Pennsylvania documentation. [126]

Phosgene is suspected to be formed by the decomposition of fluorochlorocarbons in submarine atmospheres. Webb [127] listed a maximum limit of 0.1 ppm (0.4 mg/cu m) exposure for a 90-day dive. He reported that an interim limit of 1 ppm (4 mg/cu m) for a 1-hour exposure was recommended by the Navy's Bureau of Medicine and Surgery. The limit was for a single exposure and not a permissible limit for repeated short-term exposures.

A safe concentration zone of 0.4-0.5 mg/cu m (0.1 ppm to 0.125 ppm) was recommended for international adoption in 1968 by the Joint ILO/WHO Committee on Occupational Health. [128] The Joint ILO/WHO Committee [129] prepared a survey of legislation and practices concerning permissible limits and listed MAC values for the following foreign countries:

TABLE V-1

MAXIMUM ALLOWABLE CONCENTRATION VALUES IN SEVERAL COUNTRIES

Country	Standard	
	mg/cu m	ppm
Bulgaria	0.5	0.125
Czechoslovakia	2	0.5
Czechoslovakia	4*	1*
Finland	4	1
Hungary	0.5	0.125
Poland	0.5	0.125
Rumania	0.5	0.125
United Arab Republic	4	1
Yugoslavia	0.4	0.1

* for short single exposure

From reference 129

The USSR [37] cited a permissible phosgene concentration of 0.5 mg/cu m (0.125 ppm) as established by the Main State Health Inspector of the USSR on January 10, 1959, No 279-59. If workers are in an industrial area for a brief, unspecified period, deviations are permitted with the authorization of the USSR State Health Inspectorate.

Great Britain, [130] Japan, [131] and the Federal Republic of Germany [132] based their environmental limits for phosgene of 0.1 ppm (0.4 mg/cu m) on the ACGIH value. These environmental limits were TWA concentrations for a normal working day.

The present federal standard (29 CFR 1910.1000) for phosgene is an 8-hour TWA concentration of 0.1 ppm (0.4 mg/cu m) based on the 1968 ACGIH recommendation.

Basis for the Recommended Environmental Standard

At the present time, there are no definitive data in the scientific literature concerned with long-term effects of phosgene on humans exposed at low concentrations. Human exposure to phosgene reported in the literature (see Table III-5), for the most part, consists of instances of acute overexposure, often involving a mixture of pulmonary irritants. Those epidemiologic studies [36, AF Myers, written communication, November 1974] involving exposures at low levels of phosgene suffer from a number of defects, including exposures to multiple irritants and limited analytical or medical data. In spite of the weaknesses noted, these studies are at least suggestive that the present federal standard is safe for long-term exposures. Therefore, until more conclusive evidence is developed, NIOSH recommends that the present federal standard of 0.1 ppm as a TWA concentration be maintained.

In addition, since phosgene is an acute-acting, irritant gas, it is essential to control the short-term excursions above this average. Animal experiments have demonstrated a threshold for the development of pulmonary lesions after short-term exposure to phosgene. Gross et al [42] reported

that exposure of rats to phosgene at a concentration of 3 ppm for 5 minutes resulted in slight to moderate chronic pneumonitis, while rats exposed at 1.3-1.5 ppm for 10 minutes showed no evidence of recognizable pulmonary lesions. Similarly, rat experiments conducted by Rinehart and Hatch [41] revealed that exposures to phosgene at a concentration of 1.5 ppm for 20 minutes or less did not result in any evidence of functional respiratory impairment as measured by carbon monoxide or ether uptake. In both studies, [41,42] deleterious effects were found at the lowest concentration used, 0.5 ppm, when duration of exposure was 120 minutes or greater. Although no data are available to substantiate the pulmonary effects of brief exposures to phosgene at low concentrations in humans, these studies suggest that a single exposure of 10-15 minutes' duration at concentrations of or below 1.5 ppm are likely to be safe. It is felt, however, that imposing further limitations on the degree of excursion permitted during a 10-hour workday will provide an additional margin of safety to ensure protection of the worker from the consequences of brief exposures to concentrations of phosgene above the recommended TWA limit. NIOSH is therefore proposing a ceiling limit of 0.2 ppm for any 15-minute period.

In view of the development of pulmonary edema and evidence of chronic lung changes as a result of exposure to phosgene, medical monitoring, including chest X-rays and pulmonary function tests, is required for the protection of the worker.

Professional judgment indicates that local contact with liquid phosgene is likely to cause severe tissue damage which could in part be due to the low temperature of liquid phosgene. Thus skin and eye protection, in addition to respiratory protection, is recommended for those likely to be in contact with liquid phosgene.

It is recognized that many workers are exposed to phosgene at concentrations considerably below the recommended occupational limits. Under these conditions, it should not be necessary to comply with many of the provisions of this recommended standard. However, concern for worker health requires that protective measures be instituted below the enforceable limits to ensure that exposures do not exceed the standard. For this reason, "occupational exposure to phosgene" has been defined as exposure above half the recommended TWA, thereby delineating those work situations which do not require the installation of unnecessary controls and the expenditure of health resources for provisions such as environmental and medical monitoring and associated recordkeeping.

VI. WORK PRACTICES

Strict adherence to stringent and detailed work practices is required if hazardous occupational exposures to phosgene are to be prevented. The properties of phosgene which determine to a large extent the nature of necessary work practices are: (a) the delayed and insidious onset of symptoms due to exposure to low concentrations; (b) an odor threshold which cannot be relied upon to provide an adequate warning; and (c) its irritant effects on tissue, especially the lungs. However, precautions required in the handling and usage of phosgene have much in common with those required for other irritating gases. The work practices specified in this document are derived in large part from phosgene manufacturers' literature [10,133] and the Manufacturing Chemists' Association Chemical Safety Data Sheet SD-95. [1] In addition, work practices prescribed for other irritating gases have been adapted for phosgene wherever pertinent (eg, those published in the Chlorine Manual [134]).

Warning Properties

The American Industrial Hygiene Association [90] stated that the irritant properties of phosgene were insufficient to give warning of hazardous concentrations, and that olfactory fatigue caused personnel working with phosgene to lose their ability to detect low concentrations by smell. Supporting data for these statements were not reported.

The Manufacturing Chemists' Association [1] reported that the odor of phosgene can be recognized by some persons at 0.5 ppm, but that the sense of smell is conditioned by the gas so that the odor can only be detected

briefly at the time of initial exposure. No substantiating data for these statements were cited, but many materials do have the ability to cause odor fatigue.

Leonardos et al [18] investigated the odor thresholds of 53 chemicals. They defined the odor threshold as the first concentration at which all members of a trained panel could recognize the odor of a chemical. Their tests indicated an odor threshold for phosgene of 1.0 ppm. The odor was described as being "hay-like."

In 1938, Wells et al [17] determined threshold odor detection of phosgene in a number of volunteers. Test concentrations were established with a proportioning instrument known as an osmoscope. Three tests run on 56 subjects showed that all subjects detected the odor of phosgene at or below 37.5 mg/cu m (9.4 ppm), 51.8% detected the odor of phosgene at or below 6.1 mg/cu m (1.5 ppm), and that none detected the odor below 1.8 mg/cu m (0.45 ppm).

Macy, [135] summarizing the properties and the physiological action of phosgene, stated that the threshold of odor for phosgene was 4.4 mg/cu m (1.1 ppm). Substantiating data were not included in this report.

It is concluded that the odor of phosgene, when detected, indicates the need for immediate corrective action. However, an absence of odor cannot be relied upon to indicate that exposure does not exist.

Emergencies

Personnel who have escaped from an exposure to phosgene and have inhaled dangerous quantities should be kept warm but not overheated. If possible, they should not be allowed to walk from the scene of overexposure

but should be carried. Exercise may intensify the effects of phosgene, but this has not been definitely established. In any case, physical activity should be limited to keep oxygen requirements at a minimum. Every effort must be made to treat the individual after overexposure occurs and to observe him for the onset of delayed symptoms. All facilities handling phosgene should have compressed, breathing-grade oxygen available. [1,2,10,11]

Phosgene presents no fire or explosion hazard, but high temperatures may rupture containers because of increased hydrostatic pressure. [2] In case of fire, phosgene containers should be removed to a safe place or cooled with water if phosgene is not escaping. [1]

Spills are best controlled with solutions of caustic soda or with ammonia. It has been reported [1] that some manufacturers store one ton of ammonia for each ton of stored phosgene for the purpose of neutralizing liquid spills.

Control of Airborne Phosgene

Phosgene should be used only in completely closed systems. In addition, local exhaust and general ventilation can be effective for control at points of potential escape. [1,2,10,38,58,59,90] Discharges of ventilation systems or of leaking containers may be passed through scrubbers utilizing caustic soda, ammonia, or steam to prevent phosgene from reaching the outdoor atmosphere. [1,2,90,133]

The possibility of phosgene formation must be considered when installing and operating vapor degreasers containing chlorinated hydrocarbons, since under poor operating conditions, these are potential

sources of phosgene. [5,74,136] They should be controlled to prevent exposure of operators to both solvent vapors and phosgene.

During maintenance operations on equipment in which phosgene has been present, there is a potential hazard of exposure to phosgene which is entrapped in the equipment or adsorbed on surfaces or in materials. Respiratory protection should be supplied to maintenance personnel working on equipment that has carried phosgene, unless it can be established that no phosgene is present.

Respiratory Protection

Neither chemical cartridge respirators nor half-face masks are recommended for protection against phosgene. [137] Canister-type gas masks are recommended only for rapid escape from a contaminated area because of the following limitations:

(1) Their useful life is unpredictable because of a number of variables such as breathing rate, ambient humidity, and contaminant concentration. [137]

(2) They cannot be used in atmospheres deficient in oxygen or containing phosgene in concentrations over 2.0% by volume. [137] Therefore, they are not suitable for controlling emergency exposures where the concentration of phosgene and the oxygen content of the air are unknown. [1]

(3) Users of gas masks have depended on the detection of an odor of the atmospheric contaminant to warn them of loss of protection by the canister or to indicate leaks. [1,137] However, phosgene has poor warning properties in low concentrations [1,90] and therefore the odor cannot be

depended upon to indicate a faulty gas mask in time to prevent harm to the wearer.

Training in the use of respiratory protection is required by 29 CFR 1910.134 and is stressed as an essential work practice in occupations involving potential exposure to phosgene. [1,138] Accordingly, a requirement for training is included in the standard. Recognition of the odor of phosgene should be a part of this training. [138]

Gas-mask canisters should be replaced immediately after each use, when the seal is broken, when any leakage is detected, when high breathing resistance develops, or when the recommended shelf life expires, whichever occurs first. [137]

Because of the irritant properties of phosgene, emergency respiratory protection must provide both eye and respiratory protection for the worker. Full-face masks are the only acceptable devices for employees exposed to phosgene leaks or for the protection of personnel who may be exposed to sudden high concentrations of phosgene. Masks connected to air lines or having a self-contained air or oxygen supply can also be used. Pocket- or mouthpiece-type respirators used for escape in some operations [137] are not recommended for that purpose.

Eye Protection

Liquid phosgene is probably a severe eye irritant, [11,139] as discussed in Chapter V. Protection should be provided against accidental splashes of the liquid. [90,108] Vapor concentrations of phosgene which are said to affect the eyes (1-10 ppm) are likely to affect the respiratory system as well. Accordingly, eye and respiratory protection should be combined by use of full-face respiratory protection.

Skin Protection

Liquid phosgene is said to cause severe skin burns, [1,90,139] and although this point is inadequately documented, it is a likely effect as discussed in Chapter V. Skin exposure to liquid phosgene should be prevented by the use of impervious clothing and gloves. Clothing impregnated with phosgene should be removed immediately.

Leak Detection

Continuous monitoring of areas where phosgene exposure can occur is considered essential and should be required when suitable models are available. Such monitors should be attached to alarm systems which will indicate dangerous levels of phosgene. For monitoring purposes phosgene-indicating crayons have been used. [1,10,140] Phosgene detector tubes have been described, [1,2,90] as have test papers, [2,10,90] and commercially available automatic monitor-alarms. [90, RJ Zellner, written communication, November 1974] Holding a bottle of strong ammonia water near a phosgene leak will produce a visible white cloud. [1,2,10,133] However, this should not be done unless the worker is wearing a respirator.

Leak Control

When detected, leaks should be repaired immediately by personnel wearing the proper protective clothing and respiratory protection. Leak repair kits may be assembled [1] or are commercially available. [133] Routine inspection should be scheduled to check pumps, lines, and containers for leaks; this practice is especially important during maintenance operations.[36] Leaking containers should be positioned so that

gas rather than liquid phosgene is discharged from the leak. [133] This results in less discharge of phosgene and the escaping gas cools the remaining phosgene, thus reducing pressure and leakage. Introducing moisture into phosgene can cause increased pressures sufficient to rupture containers [10,11] or can cause leaks through corrosion. [10,11]

VII. RESEARCH NEEDS

Long-term epidemiologic studies of worker populations exposed at or below the recommended environmental limits are needed. Such studies should consider not only the pulmonary effects but also the possible carcinogenic effect of phosgene. As a minimum, these studies should include environmental air measurements, medical histories, pulmonary function studies, histories of known or suspected acute exposures to phosgene, and comparisons with morbidity and mortality information for the general population.

Animal studies should be conducted to document pulmonary or other changes resulting from exposure at low levels of phosgene for extended periods. These studies should also address the issues of carcinogenicity, mutagenicity, and teratogenicity.

A sampling and analytical method for personal monitoring capable of detecting 0.05 ppm or less of phosgene should be developed. The accuracy, sensitivity, and applicability of the method to field situations should be demonstrated by a program of field and laboratory testing.

Existing methods for automatic, continuous monitoring should be tested for sensitivity, accuracy, and specificity by comparison with a reliable standard method. Since one of the primary functions of automatic, continuous monitoring is as a warning device, alarm systems should be developed and tested for reliability.

VIII. REFERENCES

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IX. APPENDIX I

AIR SAMPLING PRACTICES FOR PHOSGENE

General Requirements

Air concentrations shall be determined within the worker's breathing zone and shall meet the following criteria in order to evaluate conformance with the standard:

(a) Samples collected shall be representative of the individual worker's exposure.

(b) Sample data sheets shall include:

(1) The date and time of sample collection.

(2) Sampling duration.

(3) Volumetric flowrate of sampling.

(4) A description of the sampling location.

(5) Sampling temperature and pressure.

(6) Other pertinent information.

Breathing Zone Sampling

(a) To ensure that a sample is representative of a worker's exposure, collection shall be as near the worker's breathing zone as practical. Sampling should not hamper the typical movement patterns associated with his work.

(b) A portable, battery-operated, personal sampling pump, and a midget impinger containing 10-20 ml of a solution of 0.25% 4-(4'-nitrobenzyl)pyridine and 0.5% N-phenylbenzylamine in diethylphthalate are used to collect the sample.

(c) The sampling rate shall be 0.5-1.0 liter/minute and samples shall be taken for a time period appropriate to the estimated concentration of phosgene in the air (eg, at the recommended environmental limit, 0.1 ppm phosgene in air, a flowrate of 1 liter/minute, and a sample time of 25 minutes, 10 μg of phosgene can be collected in 10 ml of sampling reagent.)

(d) A series of short-term samples shall be taken for each TWA. The number of TWA's shall be in compliance with Table I-2 for each operation.

The TWA may be determined as follows:

$$TWA = \frac{C1t1 + C2t2 + \ldots + Cntn}{T}$$

where C = phosgene concentration during any period of time t
T = t1 + t2 +...+ tn

Sampling times should be selected so that the calculated TWA is representative of the full 8-10 hour workshift.

Calibration of Sampling Trains

Since the accuracy of an analysis can be no greater than the accuracy of the volume of air which is measured, the accurate calibration of a sampling pump is essential to the correct interpretation of the pump's indicated flow rate. The frequency of calibration should be dependent on the use, care, and handling to which the pump is subjected. In addition, the pump should be recalibrated if it has been subjected to misuse, or if it has just been repaired or received from a manufacturer. If it receives hard usage, more frequent calibration may be necessary.

Ordinarily, pumps should be calibrated in the laboratory both before they are used in the field and after they have been used to collect a large number of field samples. The accuracy of calibration is dependent on the type of instrument used as a reference. The choice of calibration instrument will depend largely upon where the calibration is to be performed. For laboratory testing, primary standards, such as a spirometer or soapbubble meter, are recommended although other standard calibrating instruments, such as a wet-test meter, or dry-gas meter, can be used. The actual setups will be similar for all instruments.

Instructions for calibration with the soapbubble meter follow. If another calibration device is selected, equivalent procedures should be used. The calibration setup for personal sampling pumps with a midget impinger is shown in Figure XIII-1.

(a) Check the voltage of the pump battery with a voltmeter to assure adequate voltage for calibration. Charge the battery if necessary.

(b) Assemble the midget impinger with the appropriate solution.

(c) Assemble the sampling train as shown in Figure XIII-1. If a prescrubber is used to remove interferents, the sampling train should be assembled with the prescrubber in line with the midget impinger.

(d) Turn the pump on and moisten the inside of the soapbubble meter by immersing the buret in the soap solution and drawing bubbles up the inside until they are able to travel the entire buret length without bursting.

(e) Adjust the pump rotameter to provide a flowrate of 1 liter/minute.

(f) Check the water manometer to ensure that the pressure drop across the sampling train does not exceed 13 inches of water (approximately 1 inch of mercury).

(g) Start a soapbubble up the buret and, with a stopwatch, measure the time it takes the bubble to move from one calibration mark to another. For a 1000-ml buret, a convenient calibration volume is 500 ml.

(h) Repeat the procedure in (g) above at least twice, average the results, and calculate the flowrate by dividing the volume between the preselected marks by the time required for the soapbubble to traverse the distance.

(i) Data for the calibration includes the volume measured, elapsed time, pressure drop, air temperature, atmospheric pressure, humidity, serial number of the pump, date, and name of the person performing the calibration.

X. APPENDIX II

ANALYTICAL METHOD FOR PHOSGENE

Principle of the Method

(a) A known volume of air is drawn through a solution of 0.25% 4-(4'-nitrobenzyl)pyridine and 0.5% N-phenylbenzylamine in diethylphthalate contained in a midget impinger. Phosgene present reacts with the solution to produce a brilliant red color.

(b) The absorbance of the sample solution is determined at 475 nm using a colorimeter or spectrophotometer.

(c) Comparison of sample absorbance with a standard curve allows quantitation of phosgene present.

Range and Sensitivity

(a) The lower limit of detection is 5 μg phosgene in the sample.

(b) The upper limit of the method may be varied, according to sample requirements, by appropriate adjustment of the usual sampling parameters (ie, sample time, solution volume, and flow rate).

(c) The 95% confidence limits for 10 degrees of freedom are ±0.007 mg at the 0.017-mg level, and ±0.003 mg at the 0.10-mg level. The precision was determined by repeated analyses by the same technician. Rather than air sampling in the field, the precision was determined by analyzing known amounts of phosgene using the same technique as the calibration procedure.

Interference

Other acid chlorides, alkyl and aryl derivatives which are substituted by active halogen atoms and sulfate esters will produce color with this reagent. However, most of these interferents can be removed in a prescrubber containing 1,1,2-trichloro-1,2,2-trifluoroethane if necessary. [104] The method is not subject to interference from carbon tetrachloride, chloroform, perchloroethylene, trichloroethylene, dichlorodifluoromethane, chlorine, hydrogen chloride, or chlorine dioxide. [85,104]

Color Stability

The red color formed is stable for at least 4 hours and should be measured within 9 hours; a color density loss of 10-15% can be expected after 8 hours. A slight decrease in color density may be expected if sampling is performed during high humidity (eg, 11% decrease at 73% relative humidity). [104]

Efficiency

Sampling efficiency is 99% or better. Evaporation losses are negligible. [85,104]

Advantages of the Method

(a) High collection efficiency.

(b) High specificity for phosgene in most likely sample environments.

(c) No elaborate equipment needed.

(d) Minimal technical expertise required for sampling and analysis.

(e) Relative ease of personal sampling.

Disadvantages of the Method

(a) Potential spillage of liquid sampling solution.

(b) Potential breakage of glass sampling equipment.

(c) Sample color instability on standing.

(d) Color density decreased by high relative humidity.

Apparatus

(a) Sampling

(1) Portable, battery-operated, personal sampling pump.

(2) Midget impinger.

(3) Flexible plastic tubing as appropriate.

(b) Analysis

(1) Glassware as required for reagent measurement.

(2) Colorimetric spectrophotometer capable of absorbance measurements at 475 nm.

(3) Spectrophotometer cells (cuvettes).

(c) Calibration

(1) Pressurized cylinder of phosgene gas.

(2) Pressure regulator.

(3) Flow meter.

(4) Appurtenant equipment for gas dilution methods or a midget impinger.

(5) Gas drying tubes.

Reagents

All reagents are ACS Reagent Grade.

(a) 4-(4'-nitrobenzyl) pyridine/N-phenyl benzylamine absorbing solution: Dissolve 0.25 g 4-(4'-nitrobenzyl)pyridine and 0.5 g N-phenylbenzylamine (may also be called N-benzylaniline) in 100 ml diethylphthalate. Store in an amber bottle.

(b) Gas purifying reagent: Approximately 50% anhydrous sodium iodide and 50% sodium thiosulfate, by weight.

(c) Diethylphthalate.

Standards

Method A: Use commercially available compressed phosgene gas (99.0%) to bubble known amounts of the gas through a midget bubbler containing the absorbing solution. Measure with a spectrophotometer (475 nm) the color developed to establish a standard curve. It has been recommended [141] that the phosgene be passed through a drying tube filled with a mixture of anhydrous sodium iodide/sodium thiosulfate prior to bubbling.

Method B: Pass the compressed phosgene gas through a tube containing anhydrous sodium iodide/sodium thiosulfate and into a glass bubbler containing diethylphthalate solution with excess normal alcoholic sodium hydroxide solution. Analyze for chloride by any of the standard titrametric methods. [142,143] Use demineralized water for this analysis. Dilute aliquots of phosgene-containing diethylphthalate with additional diethylphthalate to prepare a series of samples of known concentration (based on chloride analyses). To each sample add an equal volume of

double-strength absorbing solution (0.5% 4-(4'-nitrobenzyl) pyridine and 1.0% N-phenylbenzlamine in diethylphthalate). Mix and measure the absorbance (475 nm) after 5 minutes.

Analysis

Transfer an aliquot of the phosgene-containing absorbing solution to a spectrophotometer cuvette and measure the absorbance at 475 nm against a blank of the pure reagent. Determine the phosgene concentration from a calibration curve suitable for the specific volume of reagent used and the volume of air sampled.

Standard Curve Preparation

Using linear graph paper, plot absorbance readings on the ordinate (vertical axis) and phosgene concentration on the abscissa (horizontal axis). Standard phosgene concentrations are prepared by Method A or Method B above.

XI. APPENDIX III

PHOSGENE MONITORS

Whenever phosgene may be released or created as a result of leaks, accidents, etc, in quantities sufficient to produce exposures above the recommended ceiling concentration limit, it is essential that phosgene monitoring devices be installed, and that these devices give immediate warning of concentrations likely to be hazardous to life or health. It is difficult to define the limiting circumstances when such devices may be required, but if reasonable doubt exists, the decision should be made by a professional industrial hygienist. Monitoring devices may be based upon several operating principles, and at least one is currently (1976) commercially available. No matter what device is used, certain criteria common to phosgene monitors should be considered before purchasing any instrument.

Summary of Specifications

(a) The monitoring device must sound an alarm or otherwise warn employees whenever a phosgene concentration of 0.2 ppm is reached or exceeded.

(b) The monitoring device must have a response time of 90 seconds or less when exposed to phosgene at a concentration of 0.2 ppm.

(c) Zero drift should be less than 1% of full scale reading in 24 hours. That portion of the signal manifested as background noise should be less than plus or minus 1% of full scale reading.

(d) The monitoring device must be accurate to within plus or minus 10% of the reading for the range of 0.05-0.4 ppm phosgene.

(e) Precision and repeatability must be plus or minus 5% of full scale reading.

(f) It is desirable that the instrument respond only to phosgene, but devices which respond to other gases not normally present in the atmosphere may be acceptable. Whenever there is a possibility that gases or vapors, such as hydrogen chloride, chlorine, benzoyl chloride, acetyl chloride, and oxalyl chloride, may be present, it would be desirable to determine in advance the response of the instrument to such gases or vapors.

(g) An operating range of 0-0.4 ppm phosgene is recommended, but other ranges may be selected to suit individual needs.

(h) The device should be capable of 168 hours of continuous, unattended operation.

(i) The device and alarm should be intrinsically safe for use in locations where explosion hazards may occur.

Discussion

The principal requirements for such monitors in addition to responding to phosgene gas are that they be sufficiently rugged to withstand pressure, vibration, normal extremes of temperature, etc, and not be susceptible to plugging or interferences due to contaminants likely to be encountered in most workplaces. They should be so constructed that it is possible to routinely and quickly check the zero setting of the instrument and the response to phosgene at 0.2 ppm. It is permissible to

perform such checks by electrical means, but at least once each month instruments should be checked with a known concentration of 0.2 ppm phosgene in air.

Pressurized cylinders containing phosgene in nitrogen are commercially available. According to the National Bureau of Standards, [144] phosgene concentrations in cylinders are stable up to at least 3 months if the nitrogen originally put in the cylinder was very dry. In order to avoid the problems of degradation of the phosgene in the cylinder, it is recommended that phosgene at concentrations of 50-100 ppm in nitrogen be obtained and then diluted to the selected check concentration, ie, 0.2 ppm, as needed. This new concentration should be verified by the analytical procedure in Appendix II. Permeation tubes can also be used for generation of phosgene at known concentrations, but this requires a temperature-controlled manifold. A single calibration point at 0.2 ppm can be checked to determine whether the instrument has maintained its calibration. If the reading differs by more than 0.02 ppm from the previous calibration, then several other calibration points over the useful range of the instrument should be checked and a new calibration established. If the monitor is off by more than 0.02 ppm, the other variables should be checked, eg, flow rate, electronics, detection media.

In large plants, where there are considerable distances between work stations, additional monitors may be required to ensure worker safety. Alternately, a multipoint sampling system bringing sampled air to a single instrument may be satisfactory.

XII. APPENDIX IV

MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material shall be provided in the appropriate block of the Material Safety Data Sheet (MSDS).

The product designation is inserted in the block in the upper left corner of the first page to facilitate filing and retrieval. Print in upper case letters as large as possible. It should be printed to read upright with the sheet turned sideways. The product designation is that name or code designation which appears on the label, or by which the product is sold or known by employees. The relative numerical hazard ratings and key statements are those determined by the rules in Chapter V, Part B, of the NIOSH publication, An Identification System for Occupationally Hazardous Materials. The company identification may be printed in the upper right corner if desired.

(a) Section I. Product Identification

The manufacturer's name, address, and regular and emergency telephone numbers (including area code) are inserted in the appropriate blocks of this section. The company listed should be a source of detailed backup information on the hazards of the material(s) covered by the MSDS. The listing of suppliers or wholesale distributors is discouraged. The trade name should be the product designation or common name associated with the material. The synonyms are those commonly used for the product, especially formal chemical nomenclature. Every known chemical designation or competitor's trade name need not be listed.

(b) Section II. Hazardous Ingredients

The "materials" listed in this section shall be those substances which are part of the hazardous product covered by the MSDS and individually meet any of the criteria defining a hazardous material. Thus, one component of a multicomponent product might be listed because of its toxicity, another component because of its flammability, while a third component could be included both for its toxicity and its reactivity. Note that a MSDS for a single component product must have the name of the material repeated in this section to avoid giving the impression that there are no hazardous ingredients.

Chemical substances should be listed according to their complete name derived from a recognized system of nomenclature. Avoid using common names and general class names such as "aromatic amine," "safety solvent," or "aliphatic hydrocarbon" when the specific name is known.

The "%" may be the approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range or maximum amount, (ie, "10-40% vol" or "10% max wt") to avoid disclosure of trade secrets.

Toxic hazard data shall be stated in terms of concentration, mode of exposure or test, and animal used, ie, "100 ppm LC50-rat," "25 mg/kg LD50-skin-rabbit," "75 ppm LC man," or "permissible exposure from 29 CFR 1910.1000," or, if not available, from other sources of publications such as the American Conference of Governmental Industrial Hygienists or the American National Standards Institute Inc. Flammable or reactive data could be flash point, shock sensitivity, or other brief data indicating nature of the hazard.

(c) Section III. Physical Data

The data in this section should be for the total mixture and should include the boiling point and melting point in degrees Fahrenheit (Celsius in parentheses); vapor pressure, in conventional millimeters of mercury (mmHg); vapor density of gas or vapor (air = 1); solubility in water, in parts/hundred parts of water by weight; specific gravity (water = 1); percent volatiles (indicated if by weight or volume) at 70 degrees Fahrenheit (21.1 degrees Celsius); evaporation rate for liquids or sublimable solids, relative to butyl acetate; and appearance and odor. These data are useful for the control of toxic substances. Boiling point, vapor density, percent volatiles, vapor pressure, and evaporation are useful for designing proper ventilation equipment. This information is also useful for design and deployment of adequate fire and spill containment equipment. The appearance and odor may facilitate identification of substances stored in improperly marked containers, or when spilled.

(d) Section IV. Fire and Explosion Data

This section should contain complete fire and explosion data for the product, including flash point and autoignition temperature in degrees Fahrenheit (Celsius in parentheses); flammable limits, in percent by volume in air; suitable extinguishing media or materials; special firefighting procedures; and unusual fire and explosion hazard information. If the product presents no fire hazard, insert "NO FIRE HAZARD" on the line labeled "Extinguishing Media."

(e) Section V. Health Hazard Information

The "Health Hazard Data" should be a combined estimate of the hazard of the total product. This can be expressed as a TWA, as a permissible exposure, or by some other indication of an acceptable standard. Other data are acceptable, such as lowest LD 50, if multiple components are involved.

Under "Routes of Exposure," comments in each category should reflect the potential hazard from absorption by the route in question. Comments should indicate the severity of the effect and the basis for the statement if possible. The basis might be animal studies, analogy with similar products, or human experiences. Comments such as "yes" or "possible" are not helpful.

"Emergency and First Aid Procedures" should be written in lay language and should primarily represent first aid treatment that could be provided by paramedical personnel or individuals trained in first aid.

Information in the "Notes to Physician" section should include any special medical information which would be of assistance to an attending physician including required or recommended preplacement and periodic medical examinations, diagnostic procedures, and medical management of overexposed workers.

(f) Section VI. Reactivity Data

The comments in this section relate to safe storage and handling of hazardous, unstable substances. It is particularly important to highlight instability or incompatibility to common substances or circumstances such as water, direct sunlight, steel or copper piping, acids, alkalies, etc. "Hazardous Decomposition Products" shall include those products released

under fire conditions. It must also include dangerous products produced by aging, such as peroxides in the case of some ethers. Where applicable, shelf life should also be indicated.

(g) Section VII. Spill or Leak Procedures

Detailed procedures for cleanup and disposal should be listed with emphasis on precautions to be taken to protect workers assigned to cleanup detail. Specific neutralizing chemicals or procedures should be described in detail. Disposal methods should be explicit including proper labeling of containers holding residues and ultimate disposal methods such as "sanitary landfill," or "incineration." Warnings such as "comply with local, state, and federal antipollution ordinances" are proper but not sufficient. Specific procedures shall be identified.

(h) Section VIII. Special Protection Information

This section requires specific information. Statements such as "Yes," "No," or "If necessary" are not informative. Ventilation requirements should be specific as to type and preferred methods. Respirators shall be specified as to type and joint NIOSH and US Bureau of Mines approval class, ie, "Supplied air," "Organic vapor canister," "Suitable for dusts not more toxic than lead," etc. Protective equipment must be specified as to type and materials of construction.

(i) Section IX. Special Precautions

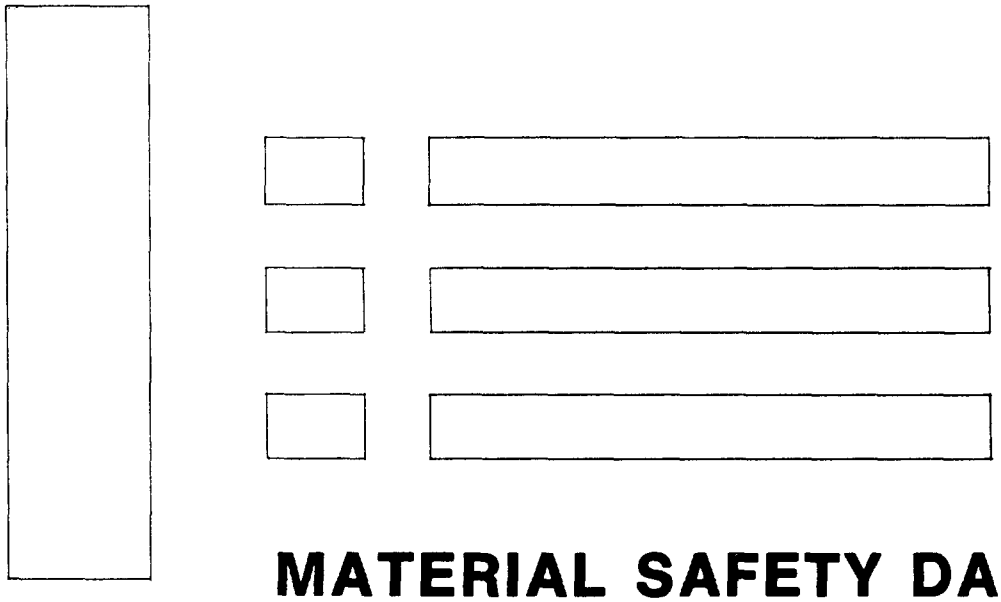
"Precautionary Statements" shall consist of the label statements selected for use on the container or placard. Additional information on any aspect of safety or health not covered in other sections should be inserted in this section. The lower block can contain references to published guides or in-house procedures for handling and storage.

Department of Transportation markings and classifications and other freight, handling, or storage requirements and environmental controls can be noted.

(j) Signature and Filing

Finally, the name and address of the responsible person who completed the MSDS and the date of completion are entered. This will facilitate correction of errors and identify a source of additional information.

The MSDS shall be filed in a location readily accessible to workers potentially exposed to the hazardous material. The MSDS can be used as a training aid and basis for discussion during safety meetings and training of new employees. It should assist management by directing attention to the need for specific control engineering, work practices, and protective measures to ensure safe handling and use of the material. It will aid the safety and health staff in planning a safe and healthful work environment and suggesting appropriate emergency procedures and sources of help in the event of harmful exposure of employees.



MATERIAL SAFETY DATA SHEET

I PRODUCT IDENTIFICATION	
MANUFACTURER'S NAME	REGULAR TELEPHONE NO. EMERGENCY TELEPHONE NO.
ADDRESS	
TRADE NAME	
SYNONYMS	

II HAZARDOUS INGREDIENTS		
MATERIAL OR COMPONENT	%	HAZARD DATA

III PHYSICAL DATA		
BOILING POINT, 760 MM HG		MELTING POINT
SPECIFIC GRAVITY (H_2O=1)		VAPOR PRESSURE
VAPOR DENSITY (AIR=1)		SOLUBILITY IN H_2O, % BY WT.
% VOLATILES BY VOL.		EVAPORATION RATE (BUTYL ACETATE=1)
APPEARANCE AND ODOR		

IV FIRE AND EXPLOSION DATA

FLASH POINT (TEST METHOD)		AUTOIGNITION TEMPERATURE	
FLAMMABLE LIMITS IN AIR, % BY VOL.	LOWER		UPPER
EXTINGUISHING MEDIA			
SPECIAL FIRE FIGHTING PROCEDURES			
UNUSUAL FIRE AND EXPLOSION HAZARD			

V HEALTH HAZARD INFORMATION

HEALTH HAZARD DATA

ROUTES OF EXPOSURE

INHALATION

SKIN CONTACT

SKIN ABSORPTION

EYE CONTACT

INGESTION

EFFECTS OF OVEREXPOSURE

ACUTE OVEREXPOSURE

CHRONIC OVEREXPOSURE

EMERGENCY AND FIRST AID PROCEDURES

EYES:

SKIN:

INHALATION:

INGESTION:

NOTES TO PHYSICIAN

VI REACTIVITY DATA

CONDITIONS CONTRIBUTING TO INSTABILITY

INCOMPATIBILITY

HAZARDOUS DECOMPOSITION PRODUCTS

CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION

VII SPILL OR LEAK PROCEDURES

STEPS TO BE TAKEN IF MATERIAL IS RELEASED OR SPILLED

NEUTRALIZING CHEMICALS

WASTE DISPOSAL METHOD

VIII SPECIAL PROTECTION INFORMATION

VENTILATION REQUIREMENTS

SPECIFIC PERSONAL PROTECTIVE EQUIPMENT

RESPIRATORY (SPECIFY IN DETAIL)

EYE

GLOVES

OTHER CLOTHING AND EQUIPMENT

IX SPECIAL PRECAUTIONS

PRECAUTIONARY
STATEMENTS

OTHER HANDLING AND
STORAGE REQUIREMENTS

PREPARED BY:

ADDRESS:

DATE:

XIII. TABLES AND FIGURE

TABLE XIII-1

CHEMICAL AND PHYSICAL PROPERTIES OF PHOSGENE

Synonyms	Carbonyl chloride, carbon oxychloride, chloroformyl chloride, CG
Chemical formula	$COCl_2$
Formula weight	98.9
Boiling point (1 atm)	7.5 C
Freezing point (1 atm)	-127.8 C
Density, liquid, 0 C	1.4187 g/ml
Density, gas, 20 C	4.39 g/liter
Specific gravity, gas, 20 C (air = 1)	3.4
Specific gravity, liquid, 19 C/4 C	1.392
Expansion ratio, liquid to gas, boiling point to 21 C	1-343
Solubility	Slightly soluble in water, hydrolyzes to hydrochloric acid and carbon dioxide. Soluble in carbon tetrachloride, chloroform, acetic acid, benzene, toluene
Color	Colorless
Odor	Sweet in low concentrations; sharp, pungent in higher concentrations
Flammability	Nonflammable

From references 1,2,10,11,12

TABLE XIII-2

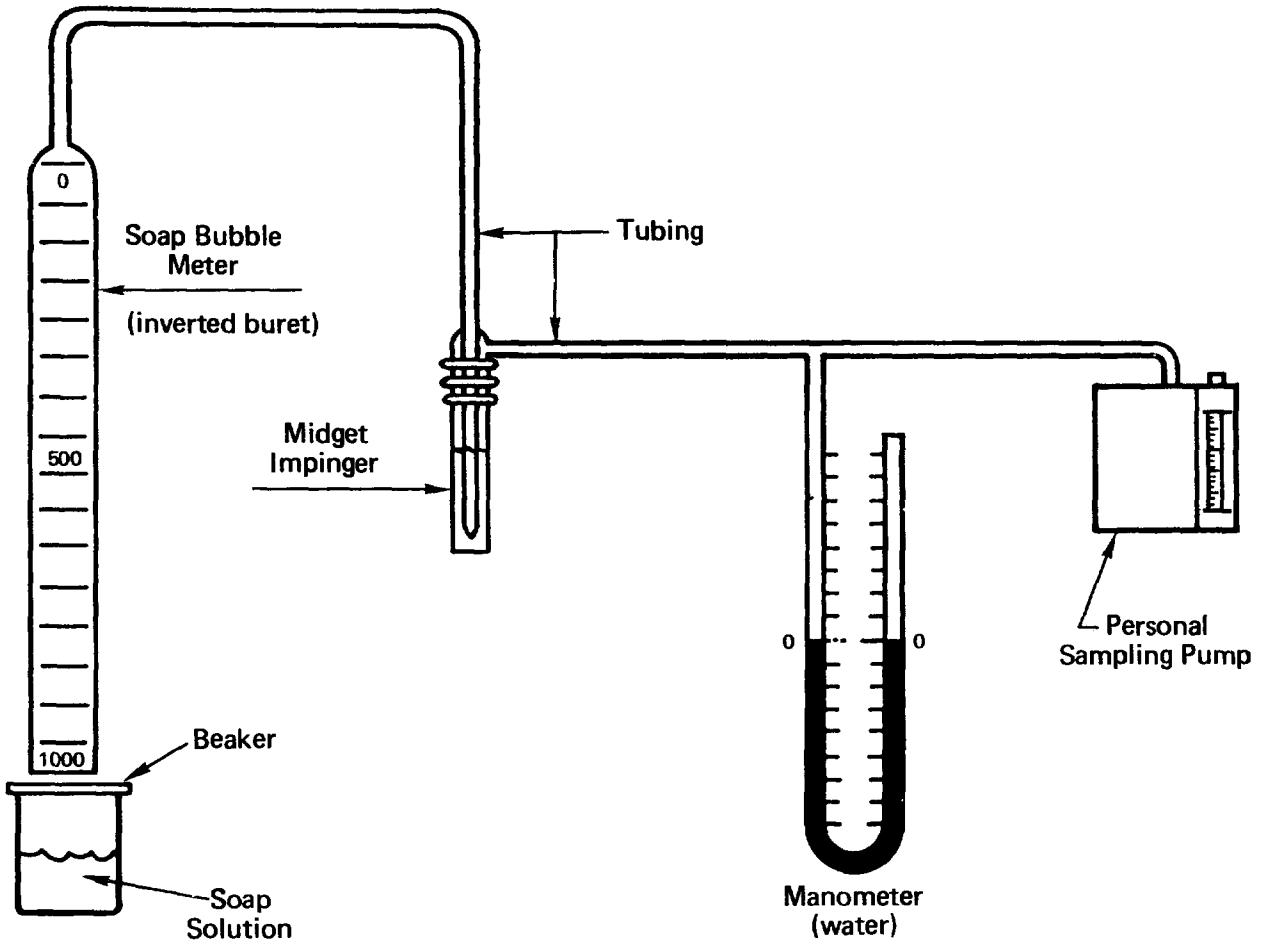
OCCUPATIONS WITH POTENTIAL EXPOSURE TO PHOSGENE

Chlorinated compound makers	Isocyanate makers
Dyemakers	Organic chemical synthesizers
Firemen	Phosgene workers
Glass makers	Resin makers
Herbicide makers	Welders or brazers (near chlorinated
Insecticide makers	solvent vapors)

Adapted from Gafafer [15]

FIGURE XIII-1

CALIBRATION SETUP FOR PERSONAL SAMPLING PUMP WITH MIDGET IMPINGER



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